

HEART-BLOCK
AND
ADAMS-STOKES DISEASE.

BY

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Heart-Block and Adams-Stokes Disease.

With especial reference to eight cases of the
Arterio - Sclerotic type observed at Walton
Workhouse Hospital, Liverpool

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PART I.

HEART-BLOCK AND ADAMS-STOKES DISEASE.

CHAPTER I.

Definitions.

Heart-Block. The stimulus for contraction passes from auricle to ventricle by the junctional system known as the auriculo-ventricular bundle. When this stimulus is stopped by some lesion or defect of this bundle the condition is known as Heart-Block.

Partial Heart-Block. The function of stimulus conveyance may be depressed and there is delay in the propagation of the stimulus, resulting in an increased a.c. interval. This depression may progress until certain stimuli fail to reach the ventricle, and the result is that certain ventricular contractions drop out; there may be only an occasional ventricular systole dropped or the ventricle may respond only to every second or third auricular systole, or even more infrequently. This partial heart-block may become complete when no stimulus reaches the ventricles from the auricles, in which case the auricles and ventricles work independently, with the result that the rhythms are entirely dissociated, the auricles pursuing a rhythm of their own, and the ventricles a slower one, the pulse in adults rarely rising above 36.

Complete Heart-Block.

Adams-Stokes Disease. Adams-Stokes Disease is a condition characterised by persistent extreme Bradycardia with syncopal, epileptiform or apoplectiform attacks, and is associated with defects in the auriculo-ventricular bundle or with lesions of the vagal nuclei or of the nerves themselves.

The occurrence of the Adams-Stokes Syndrome. This syndrome most frequently occurs when a partial heart-block is changing to a complete block (see case of Sir Wm. Gairdner): It may also occur during complete block, and is then usually associated with variations in the frequency of the ventricular systole, the pulse rate either becoming slower or more rapid both before and after the seizures, as observed by Webster and Turrell. When the conductivity of the bundle is diminished, stimulation of the vagus may induce the Adams-Stokes Syndrome.

CHAPTER II.

Short Résumé of the Clinical, Anatomical and Physiological progress.

Morgagni (1761). Morgagni was the first to give a description of the condition, but our modern knowledge of heart-block and the symptom-complex known as the Adams-Stokes Syndrome dates from the works of Adams and Stokes, two brilliant Irish clinicians, who in the Dublin chronicles first drew the attention of the medical world to this important condition. Holberton described a case in 1841.

Adams (1827).
Holberton (1841).
Stokes (1846).

Adams' patient, aet. 68, was of full habit and subject to oppression of breathing and coughing. He was first seen when recovering from the effects of an "apoplectic" attack. Dr. Adams' attention was directed to the remarkable slowness of the pulse, only thirty to the minute. The regular attendant informed Dr. Adams that, during seven years, he had seen this patient in not less than twenty "apoplectic" attacks. Death followed a subsequent attack and the post mortem disclosed a fatty heart and aortic valves studded with "specks of bone."

Stokes described the case of Edmund Butler, aet. 68, who had a permanently slow pulse and suffered from seizures which were induced by sudden exertion, distended stomach or constipated bowels. The pulse varied between 29 and 36. The duration of the seizure was seldom more than four or five minutes, and during this time the patient was quite insensible. Semibeats were heard between the contractions, weak and unattended with impulse. On second admission in June the cardiac phenomena remained as before, but a new symptom had appeared, namely, very remarkable pulsations in the right jugular vein. They were more than double the number of the manifest ventricular contractions. About every third pulsation was very strong and sudden and might be seen at a distance; the remaining waves were much less distinct, and some very minor ones could be perceived.

Stokes did not seem to understand the meaning of the semibeats, nor to have arrived at any conclusion as to the cause of the disturbance.

Charcot (1872).

Charcot in 1872 drew attention to the disease.

Chauveau (1882).

In 1882 Chauveau made observations upon a case whose pulse rate was 24 per minute. The investigations led to his publishing the following masterly description of complete heart-block: "*Ici les deux organes, oreillete et ventricule, jouaient d'une manière tout à fait indépendante. Chacun semblait travailler pour son compte sans se soucier du travail du*

voisin, et cela, d'une manière constante, permanente; jamais les deux organes ne s'influençaient réciproquement, si ce n'est quand leurs mouvements coïncidaient dans une des ces recontres fortuites que la discordance régulière des deux rythmes amenait forcément d'une manière périodique."

The cardiograms and pulse tracings given by Chauveau in proof of this assertion and his interpretation of them leave no doubt whatever that the auricles and ventricles were really acting independently of one another.

Wooldridge and
Tigerstedt (1883
and 1884).

About this time experimenters were throwing light upon the subject from physiological and anatomical standpoints. Wooldridge and Tigerstedt produced complete independence of the auricular and ventricular rhythm by physiologically separating the auricles from the ventricles.

Gaskell (1883).

Gaskell at this time was demonstrating the myogenic conduction from auricle to ventricle upon the heart of the frog and tortoise, and also demonstrated that if the auriculo-ventricular ring were clamped, as the clamp was tightened the a.v. interval was lengthened, then by further tightening a 2-1, 3-1 or 4-1 rhythm was induced. When the clamp was closed very tightly the ventricle remained still for a variable time, and then developed a rhythm of its own, or in other words a complete heart-block was induced.

In other experiments he proceeded to cut away the tissue intervening between the two chambers, and found that by gradual section the auriculo-ventricular rhythm could be altered, and when the section was complete the auricular systole had no connection with the ventricular one. He found that one part was most closely connected with this conduction of the contracting impulse, and he termed it a "bridge of tissue."

He therefore concluded that this "bridge of tissue" transmitted the auricular impulse to the ventricle and said when the bridge of "*tissue is so thin that it appears impossible to cut further, then the contraction wave is absolutely unable to pass, and the block is complete, and any contractions of the ventricles which may occur, are absolutely independent of the auricles.*"

Kent (1892).
His (1892).

In 1892 Dr. Stanley Kent and W. His, junior, discovered independently that the junctional system of the tortoise was represented in the mammalian heart by a muscular bundle, the auriculo-ventricular bundle. In March, 1892, in a paper published in the "Journal of Physiology," Kent, by cross section through the hearts of mammals, demonstrated the existence of a band of muscular tissue which preserved the integrity of the muscular connection between the two chambers of the heart, and repeated Gaskell's experiments of modifying the auriculo-ventricular rhythm by blocking this band of tissue.

In May, 1892, W. His, junior, by studying the embryo, demonstrated the existence of the muscular connection. He found that impulses passed from auricles to ventricles before any nerves had reached or were developed in the heart, and proved that the muscular connection was not broken during development, but that a connecting strand remained, which he called the auriculo-ventricular bundle. He showed its presence in man.

In 1895 he tried the effect of sectioning the bundle, and in 1899 in publishing a case showing the Adams-Stokes syndrome, he stated that he regarded the condition to be that of heart-block, and due probably to a lesion of the auriculo-ventricular bundle.

Wenckebach (1899) Wenckebach, in the same year, proved that disturbance in conduction at the auriculo-ventricular junction was the cause of certain arrhythmias.

Webster (1900). Webster in 1900 carefully studied the condition by the graphic methods. Prentiss and Edes in 1901 described the clinical features; whilst Luce and Jaquet, in 1902, stimulated physiological interest in the subject.

Hering (1905). A remarkable discovery was made by Hering in 1905. He succeeded in reviving, by perfusion, the heart of a man who had died some hours previously, and he stated that he observed nothing in his experiments on this heart which he had not already noticed in his experiments on the heart of the dog. This is strong proof of heart-block occurring in man as the result of a lesion in the bundle.

Tawara (1906). Tawara in 1906 confirmed the anatomical findings of His, and demonstrated that the fusiform cells described by Kent were Purkinje fibres, and that the bundle of His was continuous with the entire system of Purkinje fibres permeating both ventricles. He proved that the bundle of His, and the fibres of Purkinje were all parts of a great complicated system of muscular fibres which started in a well-defined node in the interauricular septum, and which connected auricle and ventricle.

Erlanger (1906). The physiological experiments of Erlanger (which will be referred to later), demonstrating the artificial production of heart-block in mammals were published about this time.

Gordon Wilson (1909). Gordon Wilson (1909), has recently demonstrated ganglion cells. By special staining methods he has demonstrated an intimate connection between the sino-ventricular connection system and branches of the vagi and sympathetic cardiac plexus.

Mackenzie (1909). In 1909 Mackenzie stated that near the base of the great veins some of the muscle remained undifferentiated, and called this tissue the sino-auricular node. Cohn, in 1910, suggested that the path of conduction between the sino-auricular node and the sino-ventricular node was by way of ordinary cardiac muscle.

Many papers have appeared within the last six years, and amongst them the cases of Barr, (1905); Hay and Moore, (1906); Gibson, (1906); Gibson and Ritchie, (1907 and 1909); Armstrong and Mönckeberg, (1911), may be described as typical of this condition.

Barr (1905). Sir James Barr (1905), published the case of the Rev. W. J. E., with history of frequently - occurring fainting attacks, accompanied by an extremely slow pulse. During the attacks of unconsciousness the pulse had fallen to 18, 15, or even 12 per minute. Auricular sounds were heard.

Seizures usually occurred when the ventricular stoppage was about 20 seconds in duration.

Tracings showed complete heart-block. The post-mortem showed that the coronary arteries near the bundle showed irregular patches of thickening; the bundle at the point of perforation was reduced to a fibrous condition and the central fibrous body was atheromatous and stretched.

Gibson (1906). Gibson in 1906 described the case of a dairyman suffering from heart-block, and the post-mortem discovered a considerable increase in the fibrous tissue of the auriculo-ventricular band, with wide separation of the muscle fibres constituting it. It is interesting to note that this was the first case in which a careful microscopic examination of the heart was made.

Hay and Moore (1906). Hay and Moore in 1906 published an excellent case showing a prolonged a.v. interval and a 2-1 rhythm. First of all the block was caused by depression of conductivity, and later this function became practically normal, and the block which persisted was found to be due to depression of excitability. Sounds were heard during the diastolic pause of the ventricles and were synchronous with the waves caused by the systole of the right auricle, and therefore it was assumed that the sounds were produced by the auricle in its systole.

Later the disease entered its second stage with syncopal, apoplectiform, or epileptiform seizures. These seizures were of all grades of severity and tended to occur in groups with periods of comparative comfort intervening.

The necropsy revealed partial obliteration of the a.v. bundle.

Gibson and Ritchie
(1907).

Gibson and Ritchie in the "Practitioner" for May, 1907, described a case of heart-block in a cabman, aet. 66. The block was at first partial and then became complete. The atropine test in this case caused the block to pass off, but as the block was known to have persisted for several months it was considered that there was some structural change in the auriculo-ventricular bundle in addition to negative dromotropic influence of the vagus.

A second case of a mason, aet. 53, whose pulse fell to 36 per minute, was also described. He was too ill to have tracings taken, and the autopsy showed that the auriculo-ventricular band passed into a mass of fibrous tissue.

Gibson and Ritchie
(1909).

In 1909, the case of Sir William Gairdner was chronicled by Gibson and Ritchie, and as the case is typical of Adams-Stokes Disease it is described in full. Sir William Gairdner had Influenza in 1899. In August of 1900 he had a series of syncopal attacks and the pulse rate was 16 per minute. In October, 1900; October and December, 1901; and in July, 1902, he had similar attacks characterised by the pulse rate falling to about 16 per minute, with unconsciousness and rigidity. Between the attacks he enjoyed fairly good health. The attacks came on at intervals until March, 1903, and after that they ceased with the exception of a transient attack in July, 1905. During these years the pulse rate varied between 28 and 32. The seizures took place when there were intermittent attacks of infrequent pulsation, and ceased when there was a persistent reduction of the pulse rate. Sounds caused by the contraction of the auricle were audible.

Graphic records of the arterial and venous pulsations and of the apex beat of the heart were obtained from time to time after the cessation of the seizures, and demonstrated complete auriculo-ventricular heart-block.

The post-mortem showed that the coronary artery was the seat of atheroma; the mitral and aortic valves were sclerosed and calcareous, and there was some fibrous transformation of the ventricular musculature. The auriculo-ventricular node and the first part of the auriculo-ventricular bundle were markedly fibrous and calcified. Calcareous deposits were found in various parts of the fibrous sheath of the bundle.

Armstrong and
Mönckeberg (1910
and 1911).

In the "Liverpool Medico-Chirurgical Journal," July, 1910, Armstrong reported a most intensely interesting case in a male child, 5½ years of age. In July, 1909, during a febrile attack of which there is no definite account, but which was probably pneumonic, Bradycardia was first discovered accidentally. During this illness he had a "long spell of unconsciousness."

A few days after becoming convalescent he "had a convulsion, lost consciousness, drew his legs up and went black in the face." He had several more similar attacks during the next and following day, and was finally recommended for admission to the Children's Infirmary, Liverpool. Whilst undergoing examination in the out-patients' department, he suddenly lost consciousness, accompanied by convulsions, general rigidity and opisthotonos. This was on August 3rd, 1909, and the case was diagnosed tuberculous meningitis. From this date until August 21st, the patient had a large number of fits, and the pulse varied between 28 and 118; on this date the pulse was 48, and a fresh diagnosis of heart-block was now ventured upon. Tracings were taken and showed complete dissociation of the auricular and ventricular rhythms. The child was put upon increasing doses of Tr. Belladonnæ, but this had not the slightest effect on his ventricular rate, which remained constantly between 36 and 48. Tracings taken in November, 1909, and in May, 1910, showed the same independence of auricular and ventricular rhythms. At this time in a critical comment on the case Armstrong wrote:—"Of course there may be some slow growing tumour which is undiagnosable."

The further history of the case is given in the "Deutschen Archiv Fur Klinische Medizin," 1911.

By a most extraordinary coincidence the boy had a second attack of Pneumonia in July, 1910, exactly a year after his first febrile attack. During this second attack the pulse was between 60 and 80, and the crisis came on the 21st July. Two days after the crisis the pulse was from 48 to 64. Next day the patient had epileptiform attacks, with pulse of 24 and 21 per minute. In the evening the pulse rose to 56. On the 25th July the boy had another syncopal attack and died in a few minutes.

Mönckeberg conducted the examination of the auriculo-ventricular bundle, and demonstrated a tumour affecting the node which he terms a lymphangio-endothelioma.

The great mass of the collected evidence—embryological, anatomical, physiological and pathological—goes to substantiate the theory that the strand of muscle which connects the auricles with the ventricles is the sole pathway by which the auricular wave of contraction passes to the ventricles.

CHAPTER III.

The Anatomy of the Sino-Auriculo-Ventricular System.

This system commences at the venous orifices in the auricles, and extends to the right auricle and thence to all parts of the ventricles, and it represents the remains of the primitive cardiac tube.

In the right auricle it commences in a well-marked ganglionic structure or reticulum known as the Knoten of Tawara. This has definite connections with the muscular wall of the right and left auricular appendages and that of the fossa ovalis and sinous venosus. Keith and Flack state that it is in connection with the sino-auricular node, the undifferentiated muscle remaining at the base of the great veins. The bundle passes over the auriculo-ventricular septum below the central fibrous body and under the septal cusp of the tricuspid valve. The main bundle is from 1 to 2 cm. long and divides at the lower part of the pars membranacea septi into right and left branches. The right branch runs along the vestigial moderator band, and gives off branches which are distributed over the surface of the ventricular wall. The left branch passes over the top of the inter-ventricular septum and broadens out into a fan-shaped layer of fibres.

The node, the bundle, and its branches, have a thin enveloping sheath of connective tissue. The main bundle is provided with a second sheath, and the space between it and the thin enveloping sheath is kept moist by fluid, and Curran suggests that this is to prevent injury to the bundle when the cardiac muscle contracts.

The nervous supply is derived from branches of the vagus and sympathetic.

The blood supply is mainly from a branch of the right coronary artery.

The Erlanger Heart-block clamp
(after Hirschfelder).



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- R.A. = Right Atricle.
R.V. = Right Ventricle.
S.M. = Septum membranaceum.
A.V.B. = Auriculo-Ventricular bundle.
A.V. = Atrial Valve.
L.V. = Left Ventricle.
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CHAPTER IV.

Experimental Physiology.

- Gaskell. As mentioned in the general survey, Gaskell's experiments on the heart of the frog and tortoise demonstrated that the auriculo-ventricular junctional band transmitted the impulse for contraction from the auricle to the ventricle (1883).
- Kent. In 1893 Stanley Kent discovered a similar band in mammals, and his experiments, using a suitably-constructed clamp, verified almost all the effects obtained by Gaskell in the frog.
- His. His, in 1895, repeated the experiments of Wooldridge and Tigerstedt, and showed that in order to bring about heart-block it was necessary to injure the auriculo-ventricular bundle only.
- Humblet. In 1904 Humblet cut the bundle in the artificially perfused heart of the dog.
- Hering. Hering, in 1905, repeated the experiments in mammals, and used a clamp which picked up a small piece of tissue containing the a.v. bundle, and by varying the pressure verified the epoch-making experiments of Gaskell, His, and Kent.
- Erlanger.
Hirschfelder.
Blackman.
Cullen. Erlanger, Erlanger and Hirschfelder, Erlanger, Blackman, and Cullen, in 1905, 1906, 1907, and 1908 further studied the physiology of heart-block in mammals. Erlanger used a special L-shaped hook of steel wire whose arm could be pressed against a brass lock by means of a bolt and screw. The hook was introduced into the right wall of the aorta just above its origin, the point passed backwards and downwards into the left ventricle and then pushed through the ventricular septum till it entered the right ventricle (*see diagram*). The brass lock was then pushed down over the long arm of the L and the nut gradually screwed taut. The following is a *résumé* of their published conclusions:—

Successive intersystolic periods lengthen until the ventricles fail to respond to one of the excitation waves. Such ventricular silences may recur with great regularity every tenth, ninth, eighth, etc. auricular beat, until alternate auricular beats fail to be followed by a ventricular contraction. The block is then partial and the rhythm is 2 to 1; upon further tightening every third, fourth, etc., auricular systole is followed by a ventricular contraction, and the 3 to 1, 4 to 1, etc., rhythms are observed. Complete dissociation of the auricles and ventricles usually follows a partial block of the 3 to 1 type, though it may follow a 2 to 1 or 4 to 1

rhythm. There are no hard and fast lines between the various degrees of heart-block. Destruction of the a.v. bundle, and of that alone, will result in complete and permanent heart-block. This makes it more than probable that in mammals, at least, conduction is muscular, but it does not conclusively prove the myogenic theory, since nerve fibres have been found ramifying through the auriculo-ventricular bundle. A dog surviving complete crushing of the a.v. bundle for a month showed complete block during the entire time.

Sudden compression of the bundle causes complete stoppage of the ventricles to occur. The ventricles cease beating, whilst the auricles continue to beat in an undisturbed fashion. The ventricles are relaxed and are gradually distended with the rhythmic injection of blood into them by the contractions of the auricles. After a pause varying in duration to 80 or more seconds the ventricles contract and empty themselves. The contractions gradually increase in frequency until the comparatively slow rate of complete block obtains. Stoppage of the ventricles is due to the more or less sudden withholdment from them of the excitation waves which have been setting their pace. Under such circumstances there seems to occur a gradual development of the inherent but dormant rhythm, so that they beat slowly at first, but with a rhythm which gradually increases to a constant but comparatively slow rate.

Stimulation of the vagus improved conductivity and facilitated the passing off of the block. When the auricular rate was increased by stimulation of the accelerators, application of heat, or rhythmic induction shocks, the degree of block was also increased.

Tabora and Hering

Tabora and Hering found that stimulation of the vagus in some cases increased the degree of heart-block and facilitated the stoppage of the ventricles.

The degree of the block depends upon the condition of the cells of the bundle of His. The graver the injury or the tighter the clamping of the bundle the lower is the irritability of the cells until finally all stimuli are refused and complete block is induced. When the heart is in a state of complete block the rate of the ventricle depends upon the irritability and rhythmicity of the ventricular musculature.

CHAPTER V.

The Syncopal, Epileptiform, or Apoplectiform Attacks.

The Seizures.

The seizures are usually due to a slowing or to a temporary stoppage of the ventricular action, causing cerebral anaemia. They occur at the passage of a normal rhythm, or of a partial heart-block to a complete heart-block; sometimes they appear during a period of complete heart-block.

The greater number of seizures appears to occur in the earlier stages when the heart-block is not complete, and the rhythm may be a 2 to 1. This was very noticeable in Sir William Gairdner's case. In this historic instance after the block became complete the ventricular rate was much more steady and less easily influenced.

Vagal stimulation may change the partial into complete heart-block, and the ventricles, being suddenly cut off from the usual rhythmic stimulus from the auricles are thrown on their own resources, and there may be a delay of many seconds before they initiate their own independent rhythm. Erlanger's experiments favour this view, as he found that complete crushing of the a.v. bundle was followed by a cessation of ventricular contractions for periods varying from a few up to 80 or 90 seconds, and that during this period the auricles continued to contract.

Stimulation of the vagus has no effect upon the ventricular rate in animals in whom the auriculo-ventricular bundle has been completely destroyed, and therefore we must look for another explanation of the stoppage of the ventricles in complete heart-block. The ventricular muscle may be ill-nourished and lose to a certain extent its irritability and excitability, and this depression at the site of stimulus production in the ventricular wall may induce ventricular stoppage. Variations in blood pressure or in the volume of the ventricular contents may influence this. The ventricle now remains silent until the stimulus matter is formed in sufficient quantity to initiate and continue the action of the ventricles.

Records have been taken showing the passage from partial to complete block without any symptoms whatsoever. In these cases it may be said that the ventricles prepare themselves by accumulating stimulus matter, and when conduction fails they are ready to initiate their own systoles at a slower rate.

CHAPTER VI.

Lesions of the Auriculo-Ventricular Node and Bundle in Cases of Heart-Block.

- Lesions.** The pathological changes may be various, the essential feature of the lesion being that it interferes with or destroys the node or bundle.
- Fibrosis and Sclerosis of the Bundle.** The majority of the cases show changes of a fibro-sclerotic nature at the auriculo-ventricular node, in the bundle of His or in the ramifications of the junctional system:—Stengel, (1905); G. A. Gibson, (1906); Hay and Moore, (1906); Aschof, (1906); Schmoll, (1906); Sir James Barr, (1906); Gibson and Ritchie, (1907); Fahr, (1907); Vaquez and Esmein, (1907); Beck and Stokes, (1908); Mönckeberg, (1908); Turrell and Gibson, (1908); Heineke, Müller, and Hoesslin, (1908); A. G. Gibson, (1908); Gerhardt, (1908); Nagayo, (1909); and Gibson and Ritchie, (1909).
- The Writer's series.** All the cases observed in the series at present under discussion almost certainly come under this category.
- Case one.** The aorta shows patches of atheroma. The coronary arteries and their branches are markedly atheromatous. A portion of the muscle ring round the mitral orifice is calcareous. On microscopic examination the bundle is obliterated from section 151-200, and is represented by dense fibrous tissue with calcareous masses anteriorly and posteriorly.
- Case two.** Shows atheroma of the aorta and of the coronary artery. There is also a large horse-shoe-shaped calcareous ring lying in the plane of the mitral valve. The pars membranacea septi is encroached upon from below by this calcareous ring.
- Case three.** The mitral valve segments present small yellowish fibrous areas in their substance. The anterior and left posterior aortic cusps are thickened, and the central portion of the right posterior cusp is calcareous. The walls of the ascending aorta and the coronary artery present nodular patches of atheroma.
- Case four.** The musculature of the left ventricle is hypertrophied and a considerable degree of fibrous change is seen in the muscle wall. The anterior and right posterior cusps of the aortic valve are slightly thickened, while the left posterior cusp is calcareous. The aortic wall presents nodular plaques of atheroma, and the walls of the coronary artery present calcareous patches of atheroma.
- Case five.** The aortic cusp of the mitral valve is markedly thick, and in its central and basal portions there are nodular calcareous masses. The basal

portions of all the aortic cusps are thick and fibrous. The ascending aorta and the coronary arteries present small patches of atheroma. Lying in the basal part of the musculature of the left ventricle, and in the plane of the mitral orifice, there is a calcareous, incomplete ring.

Case six.

The mitral valve segments are thick, shrunken, and infiltrated with calcareous deposits. A calcareous ridge projects upon the ventricular surface of the aortic cusp. The pars membranacea septi is thickened and is of small size.

The microscopic examination of the last five cases have been much delayed on account of the calcareous deposits, but the naked eye examination given in each case makes it more than probable that the lesions will be of the arterio-sclerotic type and involve the bundle.

Gummata.

A number of cases have been recorded in which the bundle has been invaded by a gumma:—Rendu, (1895); Handford, (1906); Keith and Flack, (1906); Keith, Miller and Chapman, (1906); Ashton, Morris and Lavenson, (1907); Vaquez and Esmein, (1907); Fahr, (1907); Heineke, Müller and Von Hoesslin, (1908); Robinson, (1908), and Jagie (1908).

The case of Phillips (1897), showing syphilitic disease of the heart wall may be included in this series.

Neoplasms.

Tumours of the bundle causing heart-block have been recorded. In 1892 Sandler reported a case where examination of the bundle showed a fibroma. Luce, in 1902, described a case with round-celled sarcoma of the bundle. The most interesting of this series is the case of Armstrong of Liverpool, the heart of which was examined by Mönckeberg, who found a tumour originating in the a.v. node. The tumour was a lymphangio-endothelioma.

Anæmic necrosis of the auriculo-ventricular bundle.

Anæmic necrosis of the auriculo-ventricular bundle has been described in two cases; Jellinek, Cooper and Ophüls, (1906), and MacCallum, (1907).

Fatty degeneration

In two cases, Aschof, (1906), and Butler, (1907), fatty degeneration of the bundle was found.

Ulceration.

James, (1908), described a case where a deep ulcer invaded the bundle region in the left side of the ventricular septum.

Stretching of the Bundle.

Hay published a case in 1905 in which the main lesions were (*a*) stretching of the auricular canal (the part normally within the auriculo-ventricular groove), (*b*) separation or rather attenuation of the bond between the bases of the valves, (tricuspid), and base of the ventricle.

Absence of demonstrable lesion, macro- or microscopic.	Heineke, Müller and Hoesslin published a case in which no lesion was discoverable, but where complete block had been demonstrated by graphic methods during life.
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CHAPTER VII.

Toxic Heart-Block.

Digitalis.	By the administration of digitalis Mackenzie has been able to produce partial heart-block, and states that all the cases in which he has been able to produce this condition had a previous history of rheumatic fever. If the function of conductivity is depressed and digitalis is then administered occasional ventricular systoles drop out. Mackenzie has published a tracing showing a 2 to 1 rhythm which was induced by the exhibition of digitalis to a patient who had a wide a.c. interval. Complete heart-block may be produced by digitalis in some cases of auricular fibrillation where the conductivity is previously depressed.
Pneumonia; Influenza.	Mackenzie describes cases showing partial heart-block as the result of pneumonia and of influenza.
Acute Rheumatism	May produce varying degrees of heart-block.
Typhoid Fever.	A case has been recorded by Butler in which the Bradycardia dated from an attack of typhoid fever.
Diphtheria.	Dunn describes a case in which Bradycardia and Adams-Stokes syndrome appeared on the ninth day of an attack of diphtheria.
Gonorrhœal Infection.	May be the etiological factor.
Ptomaine poisoning.	Ptomaine poisoning or absorption from the gastro-intestinal tract may give rise to the syndrome.
Streptococcic Pharyngitis and Tonsillitis.	Streptococcic pharyngitis and tonsillitis may be followed by slow pulse, with or without Adams-Stokes syndrome.

CHAPTER VIII.

Adams-Stokes Disease of Neurogenous Origin.

The Adams-Stokes Syndrome may follow a lesion of the medulla or of the vagi. In these cases there is rarely any degree of heart-block between the attacks.

Cerebral.

Fracture of the skull or meningitis may cause slow pulse and convulsions.

Atheroma of the vertebral and basilar arteries, producing sclerosis of the medulla and pons, may produce this condition (Triboulet and Gougerot).

Aneurysm, sarcoma, and gumma of the medulla may cause slow pulse and syncopal attacks. Cerebellar tumours have been recorded as causing the condition.—(Edes, Osler, Neuburger and Esinger, and Brissaud.)

Cervical.

Fracture of the cervical spine or dislocation due to injury usually causes a slow pulse alone, but pressure may cause a transient unconsciousness. Cases have been recorded where the lesion found was a narrowing of the lumen of the cervical canal.—(Osler, Holberton, and Lépine.)

Vagal.

The vagi may be attacked by a neuritis and cause this condition.—(Zurhille.)

Tanhoffer, (1875), describes a case where by compressing the vagi in the neck a colleague was able to bring on syncopal attacks.

CHAPTER IX.

The Atropine Test.

The Atropine test.

A heart-block of myogenic origin may be distinguished from one of neurogenic origin by the administration of atropine. Atropine in doses of gr. $\frac{1}{50}$ paralyzes the vagi and causes the block to pass off if it is neurogenic; it does not affect myogenic cases.

Gibson and Ritchie, in "Further Observations on Heart-Block," described a most interesting case in which both myogenic and neurogenic influences were at work. They say:

"Prior to the injection of atropine gr. $\frac{1}{50}$, the heart-block was complete and the rate of the ventricular systole was equal to 34.3 per minute. Three minutes after gr. $\frac{1}{50}$ of atropine, the block had

disappeared; each auricular systole evoked a ventricular response, the rate of contraction being 58·8 per minute. The conductivity of the auriculo-ventricular bundle was still, however, markedly lower than in health, for the a.c. interval was equal to 0·6 of a second. . . . One hour after the dose of atropine the block was again complete, and the ventricular rate had fallen to 32·7 per minute. As the administration of atropine improved the conductivity of the fibres of the auriculo-ventricular bundle, so as to lead to the transient disappearance of the block at that bundle, we are justified in concluding that, in this patient, the heart-block was at least partly due to negative dromotropic influence of the vagus. But as the heart-block was known to have persisted for several months, and to have been complete, we are confident in asserting that, in addition, there must have been some structural change in the auriculo-ventricular bundle."

CHAPTER X.

Rare instances of the Adams-Stokes syndrome.

Adams-Stokes disease may be due to recurrent groups of extra systoles causing the circulation to become so slow that cerebral anaemia is produced and syncopal attacks follow (James).

Strübing has reported the Adams-Stokes syndrome in a boy who ran for a considerable distance, and Strübing attributes the condition to cardiac overstrain.

Prentiss records a case of the syndrome following heavy lifting, in which hæmorrhage or myocardial degeneration may have been the cause of this condition.

CHAPTER XI.

The Electro-Motive Changes in Heart-Block.

In 1898, G. A. Gibson published an article embodying the conclusions he had come to in the study of the electro-motive changes produced by the systole and diastole of the heart.

At a later date he studied the electro-motive changes with especial reference to heart-block in a case whose clinical history he gave in "Bradycardia," published in 1905. The case was that of a cabman, aet. 56.

The rate of the pulse was 36, and tracings showed heart-block :—

“Simultaneous tracings of jugular and radial pulses show distinct undulations of the venous pulse between the radial pulsations, and the same may be said of the tracings obtained from the apex and the radial artery. In each cycle there is a distinct wave seen in the apex beat intermediate between the principal pulsations.”

Fluorescopy showed dissociation of auricular and ventricular contractions, the auricles beating 3 or 4 times to each ventricular systole.

The electro-motive changes were studied by means of Lippmann's capillary electrometer, and Gibson describes the electrical variations thus :—
“Leading off from the basal region of the precordia to the acid, and from the apical to the mercury, the usual diphasic movements of the capillary column were clearly seen preceding the apex beat, and evidently resulting from ventricular systole. But in the interval between these movements other smaller waves were distinctly seen, and can only be attributed to the systole of the auricles. As these latter movements were small, as observed by the direct method with the microscope, it was impossible to be certain of their exact character, but they seemed, like the ventricular, to be diphasic. When thrown upon the screen by means of the projection microscope, they were obviously diphasic in their character.”

The whole subject is now being investigated by means of the Einthoven String Galvanometer, and following Gibson, Einthoven, Lewis and Hirschfelder have demonstrated heart-block by the electro-cardiogram.

PART II.

CHAPTER XII.

CASE 1.

Complete Heart-Block with Adams-Stokes Syndrome.

THOMAS ALCOCK, Aet 62.

Date of Admission. December 11th, 1909, 4.30 p.m.

Complaint. Dizziness in the head.

General History. His occupation for 30 years was that of a baker, but during the last 15 years he has acted as a casual labourer and has been exposed to all sorts of weather, and has had several wettings. His work has always been heavy—carrying bales of cotton, hod-carrying, etc. During adult life he has consumed enormous quantities of rum and beer, to such an extent that “he could not possibly get drunk.” He has never had any accidents. There is no history of syphilis or rheumatism. He suffered from convulsions in childhood.

Two months ago he became troubled in his health. During the preceding six months he was out of work and had no proper food, on some days having no food at all. He first of all complained of feeling dizzy in the head, and suffered from noises and ringing in the ears. A fortnight before admission he fell down in the street in a faint; on getting home he had three or four more fainting attacks, and also had one or two during the night. The attacks gradually increased in number until admission, especially during the night.

State on Admission On December 11th.—Temperature 96° F.; Pulse 68; Respiration 24. Morning, December 12.—Temperature 96° F.; Pulse 44; Respiration 20. Patient seems fairly comfortable, only complaining of dizziness in the head, which he said was worse during the night than in the day. On Friday, December 17th, the patient's pulse was felt to be slower than usual, but no other signs or symptoms were noticed. On Saturday, December 18th, at 7.30 p.m., slight twitchings of the face were noticed and slight seizures lasting a few seconds. The seizures continued without intermission until death, sometimes the attacks being slight and sometimes severe, and the patient was practically in status epilepticus.

WALTON WORKHOUSE HOSPITAL.

4 HOURS CHART.

NAME *Thomas Alcock*

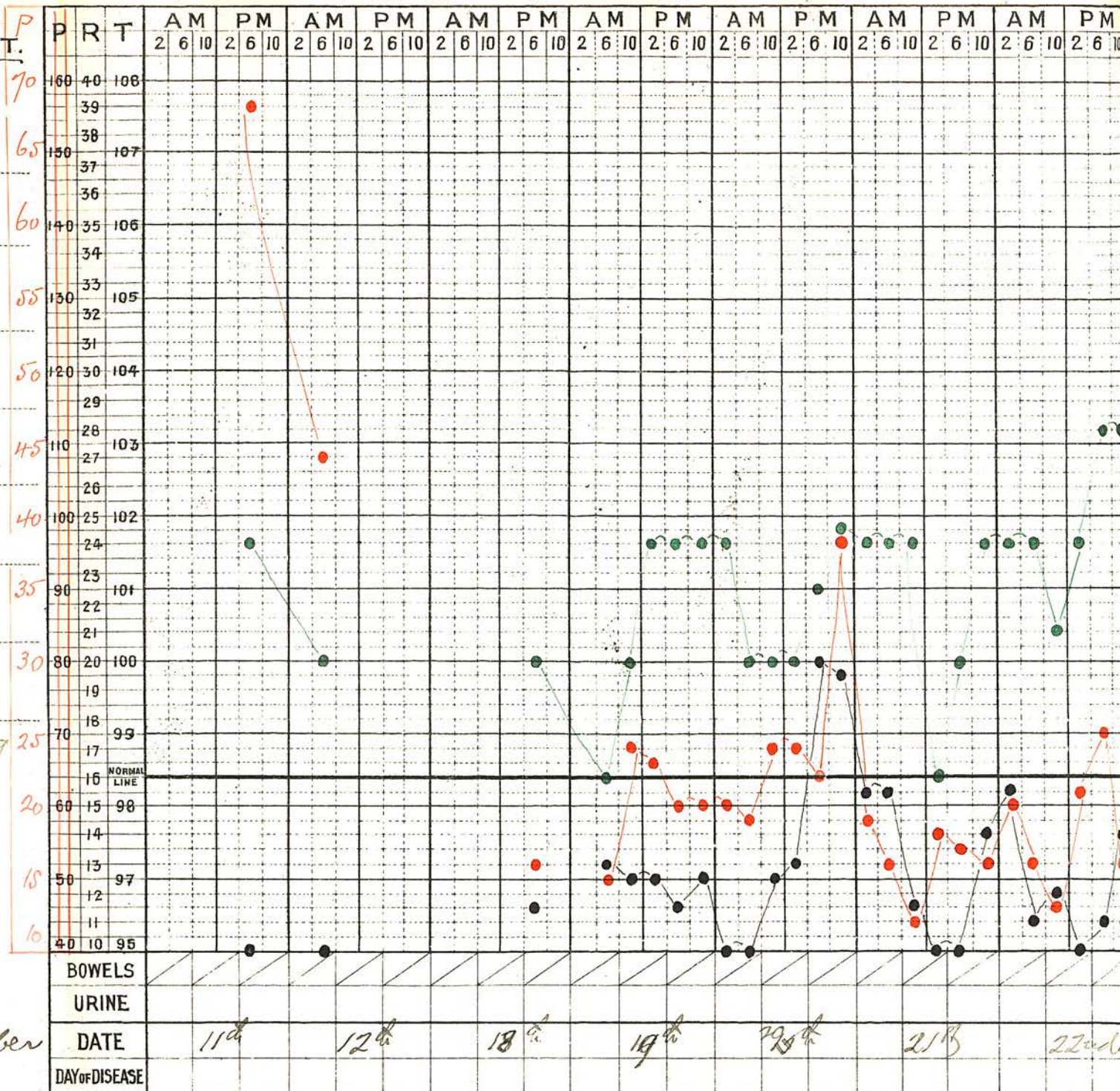
AGE *62 years*

OCCUPATION *Labourer*

ADMISSION *11th Dec^r, 1909*

DISEASE *Heart block*

RESULT *Death*
27th Dec^r, 1909



PULSE IN RED

RESPIRATION IN GREEN

TEMPERATURE IN BLACK

Circulatory system The arteries are somewhat thickened and tortuous. The systolic blood pressure is 180mm. of Hg. The pulse rate varies from 12 to 48 in frequency (*vide* pulse chart). Inspection of the neck shows visible pulsations in the cervical veins occurring much more frequently than the radial pulse. There is slight bulging of the præcordia. The apex beat is not palpable. The right border is $1\frac{1}{4}$ inches and the left $5\frac{1}{2}$ inches from mid-sternum. There is a mitral systolic bruit propagated onwards towards the axillary region. An aortic systolic murmur is present conveyed to the carotid arteries. No auricular sounds can be heard.

Nervous system. With the exception of the seizures, which will be described fully later, there is nothing of interest to note in the nervous system.

Respiratory system. There is emphysema of the lungs.

Digestive system. The liver is enlarged.

Renal system. Urine sp. gr. 1012. Reaction is acid, and there is no albumen.

Seizures. The vertiginous attacks varied in intensity and may be divided into groups.

1. *Giddiness, "Maziness," "Dying-away feeling."* He had sudden sensations as if he were about to fall.
2. *Transient loss of consciousness* with pallor, but without loss of control of the voluntary muscles, a true "petit mal."
3. *Transient loss of consciousness with slight rigidity and twitchings, limited to the face and arms.*
4. *Generalised convulsions with loss of consciousness*, showing all the characteristics of a true epileptic attack.
5. *Syncopal attacks.* The patient suddenly becomes unconscious, face is pale or ashen grey in hue, with absence of rigidity and twitchings.

The patient had hundreds of these attacks, mostly of the milder type. From the 26th December until his death the attacks were much less frequent and were more syncopal in nature.

Description of a seizure. The first thing that is noticed is the sudden arrest of the radial pulse for about 12 seconds. He then looks pale and dazed, cannot speak, but hears what is said to him. He slowly rises a little from the bed, his eyes staring fixedly in front of him and his limbs rigid. Consciousness is now entirely lost and he falls back in a convulsion, breathes stertorously and

deeply, becomes cyanosed, grimaces, throws his legs and arms about and picks at the bed-clothes. The convulsive movements cease, he suddenly regains consciousness and looks round in a wild alarmed manner. He is mentally confused after the attack. There is now a gradual acceleration of the ventricle to its usual slow rate after the convulsion is over.

Aura.

He states that before many of the attacks he sees a bright white ball of light on the ceiling.

Observations.

Observations, each of half an hour's duration, were made, and the numbers of seizures taking place, with any additional peculiarities, are noted in the tables which appear on next and subsequent pages.

TABLES OF OBSERVATIONS MADE.

Observation.	Date when made.	Duration of Observation.	Number of Seizures Occurring.	Duration of Seizure.		Aggregate Time of all Seizures.	
				Shortest.	Longest.		
First	Dec. 22	Minutes. 30	20	Seconds. 2	Seconds. 15	Min. 2	Sec. 37
Second	23	30	21	8	28	5	9
Third	24 Morning	30	13	7	23	2	54
Fourth	24 Evening	30	22	4	19	4	8
Fifth	25	30	10	Not recorded.			
Sixth	26 Morning	30	9	5	20	1	37
Seventh	26 Evening	30	6	4	13	0	48

Observation.	Seizure.	Duration.	REMARKS.
First. Made Dec. 22	1st	Seconds 10	
	2nd	10	
	3rd	15	
	4th	8	
	5th	2	
	6th	12	
	7th	8	
	8th	7	
	9th	7	
	10th	5	
	11th	8	
	12th	8	
	13th	7	
	14th	4	
	15th	6	Plucking at bedclothes ; semi-conscious.
	16th	7	
	17th	4	
	18th	9	
	19th	5	Pulled and violently picked the bedclothes.
	20th	15	

TABLES OF OBSERVATIONS—*Continued.*

Observation.	Seizure.	Duration.	REMARKS.
Second. Made Dec. 23rd.	1st	Seconds 20	Threw arms about and grimaced.
	2nd	10	
	3rd	9	Slight.
	4th	8	
	5th	10	More severe.
	6th	11	
	7th	13	More severe; threw legs and arms about.
	8th	28	
	9th	9	Very severe.
	10th	23	
	11th	22	Slight; plucking at bedclothes.
	12th	20	
	13th	12	Violently jerked arms and legs.
	14th	8	
	15th	15	
	16th	23	
	17th	8	
	18th	18	
	19th	19	
	20th	11	
	21st	12	
Third. Made Dec. 24th (Morning).	1st	Seconds 15	Threw arms about and grimaced.
	2nd	13	Slight; picked at bedclothes.
	3rd	10	
	4th	23	More severe; threw arms and legs about.
	5th	15	Slight.
	6th	18	Rather severe.
	7th	7	Threw arms and legs about.
	8th	13	
	9th	15	Rather severe.
	10th	8	Very slight.
	11th	10	Moved arms and legs.
	12th	12	Grimaced.
	13th	15	Breathing was stertorous and heavy during all these seizures.

TABLES OF OBSERVATIONS—*Continued.*

Observation.	Seizure.	Duration.	Remarks.
Fourth Made Dec. 24th (Evening).		Seconds	
	1st	12	
	2nd	8	Rather severe.
	3rd	18	
	4th	8	Arms and legs jerked.
	5th	5	Slight.
	6th	12	
	7th	14	Slight.
	8th	10	Threw arms and legs about.
	9th	8	Grimaced and plucked the bedclothes.
	10th	16	
	11th	13	
	12th	9	
	13th	9	Jerked right arm.
	14th	13	
	15th	4	Very slight ; did not lose consciousness.
	16th	15	
	17th	19	More severe ; jerked arms and legs.
	18th	13	Severe.
	19th	8	Threw both arms above his head.
	20th	5	Slight.
	21st	13	
	22nd	16	
Fifth Made Dec. 25th.	1st to 10th	Seconds	
		} Not recorded.	The convulsive movements were much less marked.
Sixth Made Dec. 26th (Morning).		Seconds	
	1st	20	No movements.
	2nd	12	Very slight movements.
	3rd	13	
	4th	10	Slight movement of arms.
	5th	5	
	6th	6	
	7th	8	
	8th	12	
	9th	11	
			Seizures are syncopal in nature ; breathing is less stertorous.

TABLES OF OBSERVATIONS—*Continued.*

Observation.	Seizure.	Duration.	REMARKS.
Seventh. Made Dec. 26th (Evening).	1st 2nd 3rd 4th 5th 6th	5 13 9 12 5 4	} Dozing most of the time; fits are purely syncopal in nature.
Dec. 27th.	1st 2nd	45 75	Prolonged syncopal attack. Ditto ditto

The number of seizures occurring during the 7 half-hourly observations, made from 22nd to the 26th December, was ~~121~~, equal to 17.3 per half-hour, and as he continued having the seizures between the observation periods at about the same frequency, the average number per diem was approximately 830.

Progress and treatment of the case. On admission treatment was symptomatic and he was put upon Easton's syrup and potassium bromide. His diet was light and plain.

When the seizures commenced atropine in doses of gr. $\frac{1}{100}$ was given every four hours, but was discontinued in a few days as there was no change in the pulse rate. Vagus stimulations (pressure) were tried without any result. He was now put upon a mixture containing potassium iodide grs. xv. and sodium nitrite grs. ii. three times daily. Brandy half an ounce four hourly was also administered.

The seizures as above described became more syncopal in nature (pallor, loss of consciousness, with absence of rigidity and twitching), the last one being of 75 seconds duration. He died at 9.45 p.m. on December 27.

REMARKS ON TRACINGS.

The tracings are in four separate records numbered 1, 2, 3, 4. They constitute one continuous tracing and were cut up to facilitate examination and analysis. These demonstrate six seizures labelled A, B, C, D, E, F. The characteristic features of each seizure is described in detail. The records will be found in separate compartments in the accompanying box, labelled "Tracings of Thomas Alcock."

RECORD No. (1)

No. 1 is a record of one complete fit preceded by a period of frequent ventricular action, and followed by a similar period. 15 seconds duration of ventricular stoppage before the onset of convulsive movements; 33 to 34 seconds before the ventricle begins to contract again. Immediately preceding ventricular stoppage the time elapsing between two consecutive auricular systoles is about $5\frac{1}{3}$ fifths of a second. With the cessation of ventricular systole the auricular frequency rapidly increases; 8 seconds after the cessation of ventricular systole the tracing shows only $3\frac{1}{4}$ fifths of a second between two auricular systoles. When the convulsive period of the seizure began it was not possible to record the venous pulse owing to the movements of the patient. A record of the jugular pulse was again obtained 13 seconds after the first appearance of the radial pulse as shown by the tracing. The auricles are found now to be beating even more frequently, an auricular systole occurring every 3 fifths of a second, and a few seconds later every $2\frac{5}{8}$ fifths. As will be seen from the tracing, from this point on the auricular frequency steadily diminishes until the auricular systole occurs about every second.

In the first portion of this record the auricle is beating every 3 fifths of a second. On one occasion the interval was $2\frac{3}{4}$ fifths. Gradually the frequency diminishes, and during the 15 seconds actually preceding ventricular stoppage the auricle beats at intervals of about 1 second. Immediately preceding the ventricular stoppage the intervals are $5\frac{1}{3}$ fifths.

The following points become clear :—

Ventricular stoppage is preceded by marked slowing of the auricle, and is at once followed by a rapid increase in auricular frequency. This increase in the auricular frequency continues in a somewhat marked degree for some time after the ventricle has resumed its activity, and then gradually the auricle begins to slow down.

When it reaches a frequency of about 60 per minute another seizure is imminent.

Tracing (1) begins shortly after a period of ventricular stoppage.

RECORD No. (2).

This record represents two distinct periods of ventricular stoppage, B and C, similar to that described in record (1).

The tracing begins with the auricle beating every 3 fifths of a second, and the time between two consecutive ventricular systoles is 13 fifths; the

auricle gradually decreases in frequency until the time between two consecutive auricular systoles is $5\frac{1}{2}$ or even 6 fifths of a second. This rate just precedes the seizure B. Simultaneously the period between two consecutive ventricular beats is 6 fifths of a second. The auricle is then beating at almost the same rate as the ventricle, though quite independently, as a careful analysis of the jugular record shows.

The seizure B is similar to that already described on record (1). There is the same ventricular stoppage, followed by the same rapid increase in auricular frequency. The period of complete ventricular stoppage is of at least 30 seconds duration, the rigidity and convulsive efforts beginning at the end of 13 or 14 seconds. The first contractions of the ventricle are very infrequent, a ventricular systole occurring about every 5 seconds, and at the same time the auricle is still beating with great frequency—every $2\frac{5}{8}$ fifths of a second. The ventricular rate gradually increases until the period between consecutive systoles reaches a little over a second. By this time the auricle has slowed down and its frequency approximates to that of the ventricle (see diagram). Then another seizure takes place similar in most respects to those already described; the rigidity and convulsions began 15 seconds after the onset of ventricular stoppage (see record.)

RECORD No. (3).

Another beautiful example of one complete seizure (D); an exceptionally good jugular tracing during the first 15 or 16 seconds of the attack. The characteristics are much the same as those already described, the last ventricular cycle preceding the ventricular stoppage being of $6\frac{1}{3}$ fifths duration; at the same time the auricular cycle was 6 fifths. The stertorous breathing and convulsive movements are well shown in the record. The ventricle resumes its activity at a rate of about 12 beats per minute, and gradually increases in frequency; the length of the auricular cycle remains about $3\frac{1}{4}$ fifths up to the end of the record.

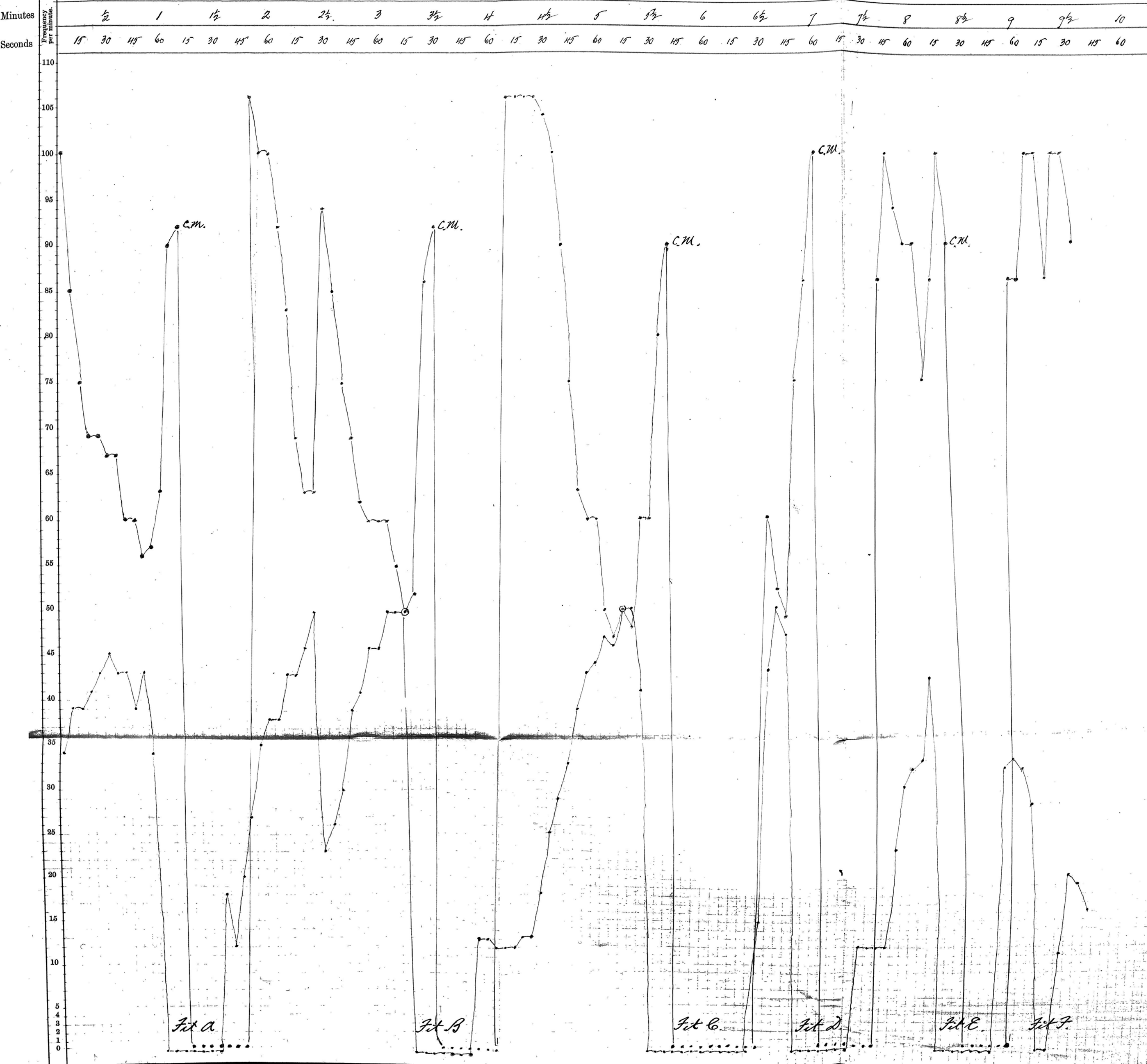
RECORD No. (4).

The 4th record begins 2 or 3 seconds before the onset of a period of ventricular stoppage (E), and this seizure is characterised by the features already described. Its termination is, however, slightly different to that of seizure A, B, C and D, because here the ventricle at once begins to beat at a more frequent rate than in the other seizures. Each ventricular cycle is only 9 or 10 fifths of a second. After 10 such contractions there is another period of ventricular stoppage. This time the duration of the stoppage is only 11 seconds, and it is interesting to note that there was no

C.M.=Convulsive Movements.

Thomas Alcock.

Chart to show the relationship of the Auricular and Ventricular frequency to the Seizures.



THOMAS ALCOCK.

Prints a1, a2, a3, a4, a4—, a5, a6, a7 and a8.

THE FIGURES INDICATE:—

- 1 Right Auricle
- 2 Superior Vena Cava
- 3 Thebesian Valve at mouth of Inferior Vena Cava
- 4 Fossa Ovalis
- 5 Mouth of Coronary Sinus
- 6 Marginal Cusp of Tricuspid Valve
- 7 Septal Cusp of Tricuspid Valve
- 8 Aorta
- 9 Pulmonary Valve
- 10 Left Auricle Collapsed
- 11 Aortic Cusp of Mitral Valve
- 12 Marginal Cusp of Mitral Valve
- 13 Left Auricular Appendix
- 14 Branches of Left Coronary Artery
- 15 Coronary Sinus
- 16 Pulmonary Artery
- 17 Infundibular Cusp of Tricuspid Valve
- 18 Right Posterior Aortic Cusp
- 19 Anterior Aortic Cusp
- 20 The Area of Calcification

A 1. Inner or Septal aspect of the Right Heart.



THOMAS ALCOCK

A 2. Outer aspect of the Left Heart.



THOMAS ALCOCK



A 3. Inner aspect of the Left Heart.

THOMAS ALCOCK



A 4. Same as A 3, but with rather more of the Ventricular wall cut away to expose the Aortic Orifice at the top left-hand corner of the triangle formed by the Ventricular cavity, and with the Aortic Cusp of the Mitral Valve turned back or up so as to show the position of the calcareous mass in relation to the valve.

A 4—, Right Heart after the removal of the Block for sections.



THOMAS ALCOCK

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A 5, A 6, A 7 and A 8. A 5 shows the Block from its posterior aspect mainly A 6, Right and posterior aspect. A 7 and A 8, Left and anterior aspects. In 8 the Aortic Cusp of the Mitral Valve is in natural position, whereas in 7 it is held backwards so as to expose the calcareous ridge.

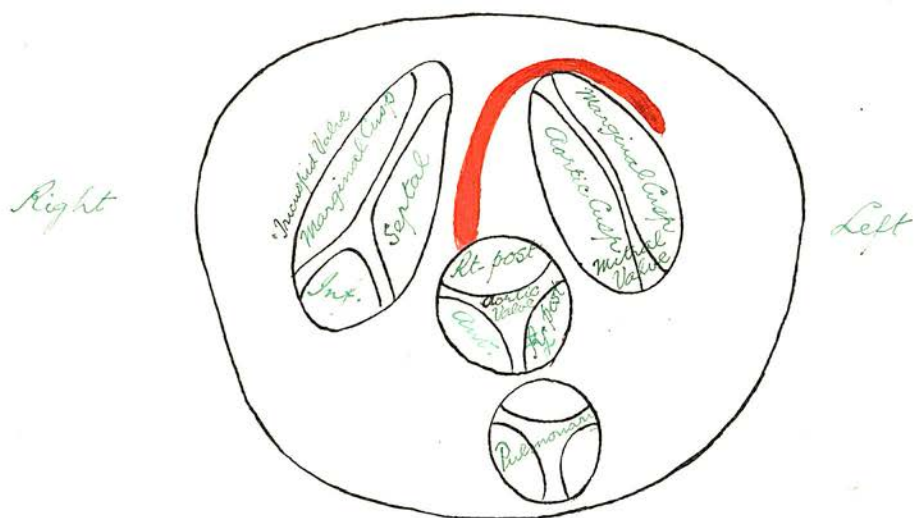
rigidity or convulsions during or after this phase, the cerebral anæmia not having been sufficiently prolonged. Preceding the seizure (F), the auricular frequency has not slowed down to anything like the same extent as seen in the other records. What the relationship is between the auricular rate and the shortness of the period of ventricular stoppage it is difficult to say. This is the only seizure of the 5 of this character, and it follows on an attack in which the ventricle resumes its contractions at a rapid rate, and one notices that the auricular frequency is maintained up to the period of ventricular stoppage. The graphic chart of the auricular and ventricular frequencies shows the intensely interesting manner in which the frequencies approximate before a seizure (see chart).

The schematic diagram constructed on Record No. 2 after fit (B) shows complete auricular-ventricular heart-block.

Pathological Examination of the Heart.

Heart.	The heart weighs 460 grammes.
External measurements.	Circumferential measurements in the long axis of the heart is 37cm. and around the auriculo-ventricular groove 29.3cm.; length, 14.6cm.; breadth, 11.2cm.; antero-posteriorly, 8.4cm.
Internal diameters of cavities.	Right auricle, 6.7cm.; right ventricle, 7.0cm.; left auricle, 6.1cm.; left ventricle, 6.5cm.
The epicardium.	The epicardium is thick over both auricles, and over the basal part of the ventricles posteriorly epicardial fat is scanty, except over the apical portion of the right ventricle where the layer of adipose tissue is 5mm. thick.
Right auricle.	The endocardium presents slight, diffuse thickening. The foramen ovale is closed. The musculature is not hypertrophied. No lesion can be seen with the naked eye at the junction of the superior vena cava and auricle.
Tricuspid orifice.	The tricuspid orifice is 12cm. in circumference. The valve segments are slightly thickened but not calcareous. The chordæ tendineæ are healthy.
Right ventricle.	The endocardium shows slight, diffuse thickening. The columnæ carneæ are moderately hypertrophied. No fibrous change is visible.
Pulmonary orifice.	The pulmonary orifice is 8cm. in circumference. The pulmonary valve and artery are healthy.

Thomas Alcock



The Calcareous ring in red.

- Left auricle. The endocardium presents marked diffuse thickening. The musculature is slightly hypertrophied.
- Mitral orifice. The mitral orifice is 8cm. in circumference. The marginal cusp is not thick or shrunken.
- The aortic cusp is thickened but not shrunken. The chordæ tendineæ are healthy. The papillary muscles are hypertrophied but not fibrous.
- Left ventricle. The muscle wall at the apex of the chamber is 1.8cm. thick; mid-way between the apex and the base of the ventricle the thickness of the wall is likewise 1.8cm. The columnæ carneæ are somewhat hypertrophied, but no fibrous change is seen in the ventricular musculature.
- The aortic orifice. The aortic orifice is 7.2cm. in circumference. The cusps are thickened but not shrunken or calcareous. The wall of the aortic arch presents a few nodular patches of atheroma, with pultaceous, but not calcareous, material in their deeper parts. The coronary arteries and their branches are markedly atheromatous.
- The calcareous ring. A portion of the muscle ring round the mitral orifice is calcareous. The calcareous mass lies in the subendocardial layer of the muscle close under, but not involving, the marginal cusp of the mitral valve. The mass commences a little internal to the mid-point of the line of attachment of this cusp, and passing inwards in the basal part of the ventricular muscle it is represented in the photographs (Fig. 20). At this point the transverse section of the calcareous ring measures 7mm. by 4mm. Thereafter the calcareous ring passes in the muscle under the line of attachment of the aortic cusp of the mitral valve (see photograph 4), and then passes forwards in the ventricular septum, forming a prominent ridge a little below the attached margin of the right-posterior cusp of the aortic valve (see photograph). The calcareous mass terminates below the mid-point of the line of attachment of this cusp.

Examination of the Auriculo-Ventricular Bundle.

The sections are all cut in the same plane which is roughly parallel to the upper level of the segments of the aortic valve. The node is fibrous and so is the bundle. Many calcareous masses are in close proximity to the bundle, and some encroach on it.

The extensive calcareous deposits behind the node and above it are not of much significance, for the node seems always in good connection with the musculature of the right auricle, *i.e.*, along the septal wall of the right auricle.



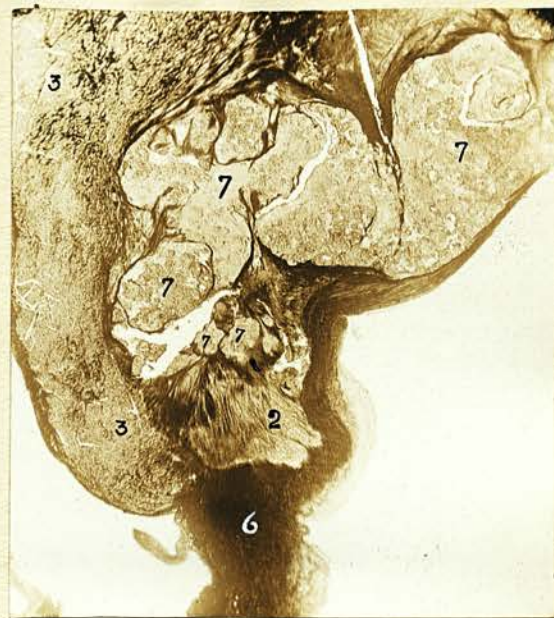
I



II



III



IV

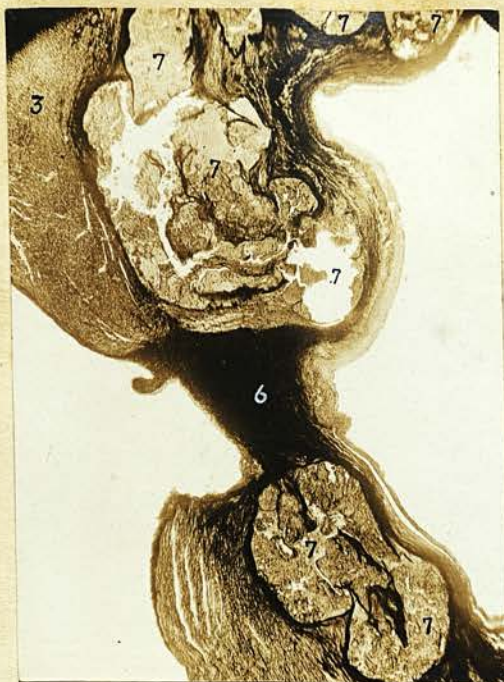
THOMAS ALCOCK.

- I. Photo-micrograph of section No. 79, $\times 11.5$
- II. Photo-micrograph of section No. 97, $\times 11.5$
- III. Photo-micrograph of section No. 119 $\times 11.5$
- IV. Photo-micrograph of section No. 145. $\times 11.5$

1, The auriculo-ventricular node; 2, The auriculo-ventricular bundle; 3, Ventricular musculature; 4, Auricular musculature; 5, Central fibrous body; 6, Pars membranacea septi; 7, Calcareous nodule; 8, Blood vessel.



I



II



III

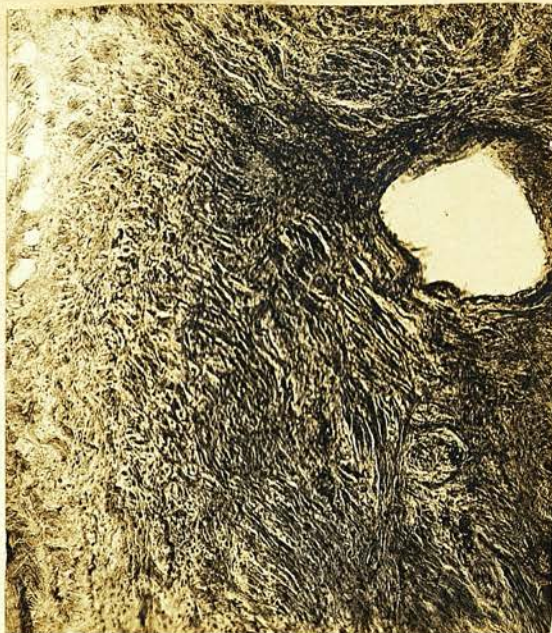


IV

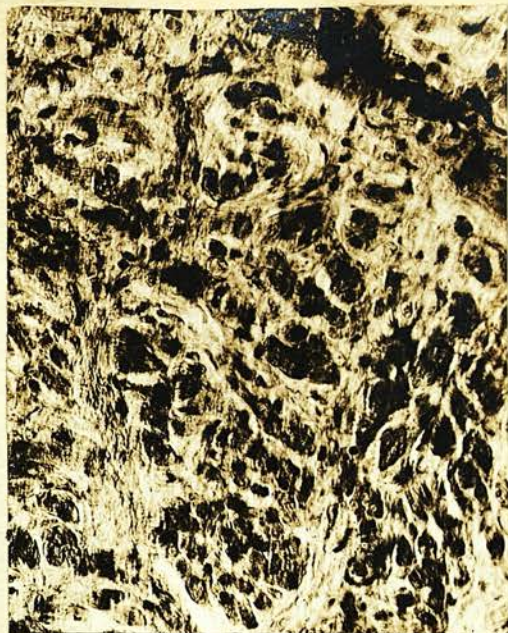
THOMAS ALCOCK.

I, II, III and IV, photo-micrographs of sections 157, 167, 197 and 219, all $\times 11.5$

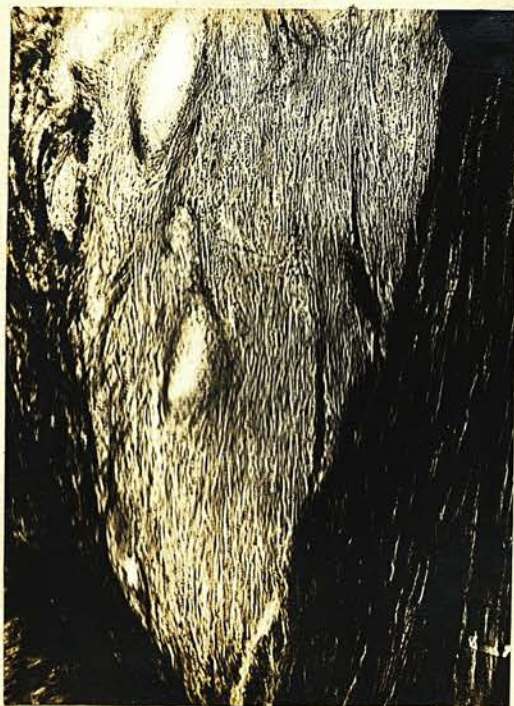
2, The auriculo-ventricular bundle; 3, Ventricular musculature;
 4, Auricular musculature; 6, Pars membranacea septi;
 7, Calcareous nodule; 9, Septal cusp of tricuspid valve.



I



II



III



IV

THOMAS ALCOCK.

I. Micro-photograph of section 83, $\times 75$; shewing the auriculo-ventricular node with its artery, and the commencement of the auriculo-ventricular bundle.

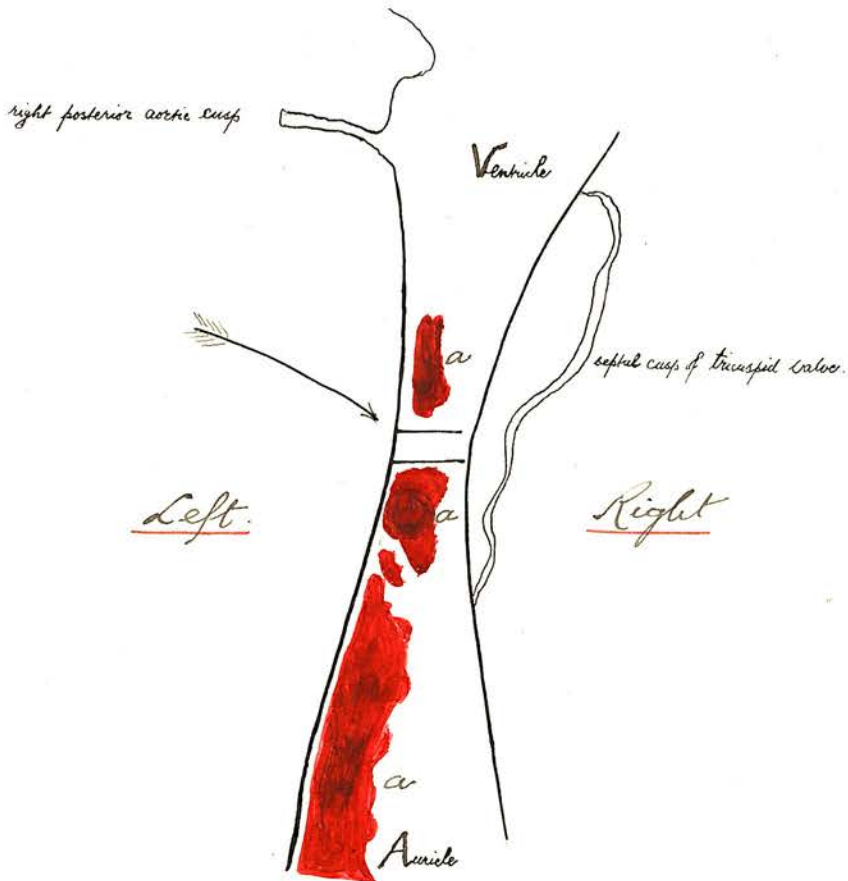
II. Photo-micrograph of section 83, $\times 410$; shewing diffuse fibrosis of the auriculo-ventricular node.

III. Photo-micrograph of section 83, $\times 75$. The auriculo-ventricular bundle is passing forwards through the central fibrous body, and is infiltrated with leucocytes.

IV. Photo-micrograph of section 117, $\times 75$; shewing increase of fibrous tissue and leucocytic infiltration of the a - v bundle.

Thomas Alcock.

Diagram of Calcareous deposits,
seen in sections 160 to 180.



a = Calcareous masses

The arrow points to the dense fibrous
tissue of pars membranacea septi.

Nodal tissue appears in sections Nos. 55 and 57. By section 71 the bundle is visible. In section 125 the bundle is starting to divide, and in section 151 is just above the lowest part of the pars membranacea septi. The bundle becomes wholly lost about sections 157 to 175 or 180, in dense fibrous tissue at the very lowermost part of the pars membranacea septi, with large calcareous masses on the posterior side of the pars membranacea septi—where the bundle ought to be seen dividing, or just divided—and also calcareous masses anteriorly below the right posterior aortic cusp. The calcareous deposits are even more marked in sections 160 to 180. About section 197 or 200, the left branch of the bundle may be picked up, but from section 151 to 200 there seems to be no remains of the bundle at all. The case must, therefore, be considered to be one of complete heart-block, anatomically speaking.

Naked Eye and Microscopic Examination of the Brain and Vagus Nerves.

The brain.

The brain, after fixation in formalin, weighs 1,400 grammes. To the naked eye it appears normal in shape, and the convolutions are well defined and the meninges look natural. There does not seem to be any asymmetry the result of local atrophy of any part. The veins over the cortex appear natural, but there is definite, though slight, atheroma of the vessels at the base. This atheroma is by no means universal, however, considerable lengths of the various branches of the circle of Willis being free from it. The most markedly thickened patches are in the basilar artery, in the left internal carotid, and in the commencement of the left middle cerebral artery.

Medulla oblongata.

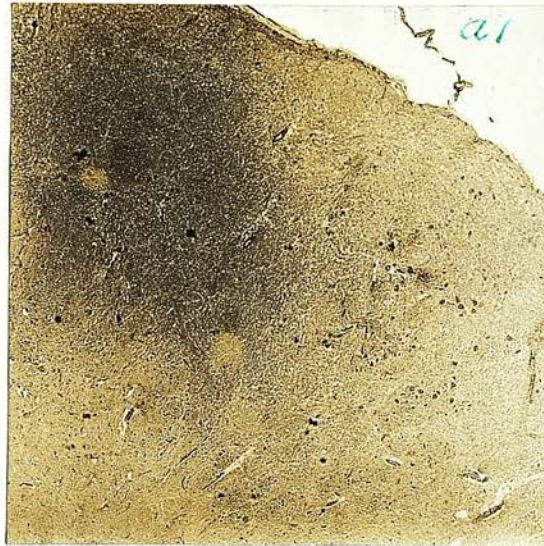
The medulla, like the cerebrum, looks natural to naked eye.

On cutting into the substance of the brain, pons, medulla, and cerebellum, no microscopic abnormality is to be detected at any part; the structural definition of the different parts is well defined; there is no area of softening to be noticed near either the cortex, internal capsule, or in the pons, or medulla. There is no hæmorrhage or blood extravasation. The lateral ventricles and the third ventricle are not dilated, and the fourth ventricle, with its choroid plexuses, seems natural. In short, to the naked eye there is no abnormality to be recorded beyond a very slight degree of atheroma of some of the vessels at the base.

Vagal nuclei.

The two chief abnormalities to be noted in section through the medulla oblongata at the level of the vagus nuclei are, first, that the total number of nerve cells to be seen in the vagus nuclei is relatively small, as though some had disappeared entirely, and secondarily, that although some

THOMAS ALCOCK



× 24 Diameters

A 1. Transverse section through Medulla Oblongata
at level of the Vagal Nuclei.

THOMAS ALCOCK



× 24 Diameters

A 2. Section of the Basilar Artery.

THOMAS ALCOCK



× 15 Diameters

A 3. Section from the immediate branches of the
Internal Carotid Artery.

THOMAS ALCOCK



× 33 Diameters

A 4. Transverse section of Vagus Nerve.

of the nerve cells stain well and show well-differentiated protoplasmic granules, nucleus and nucleolus, others are in what appears to be a degenerative state, without nucleus or nucleolus and without any clearly defined protoplasmic granules. There is no small round-celled infiltration nor any excess of leucocytes in the pericellular or perivascular lymph spaces.

Basilar artery. Section taken from the basilar artery shows a practically normal vessel, the slight atheroma that was thought to be present on naked eye examination not being confirmed microscopically.

Internal carotid artery. Section from the immediate branches of the internal carotid artery also shows no decided evidence of atheroma or other abnormality microscopically.

Vagus nerves. Section shows no excess of perineural or endoneural connective tissue, no evidence of small round-celled infiltration, and no vascular engorgement. There are an unusual number of hyaline bodies present, such as are apt to take the place of degenerate nerve tissues; but otherwise no definite abnormality, other than changes which are due to post mortem alterations, can be detected. The elongated cleft-like spaces in the section are almost certainly artificial. There are a large number of apparently empty nerve sheaths and it is possible that they were empty ante-mortem as the result of degeneration of the nerve fibres within them.

CASE 2.

Complete Heart-Block with History of Adams-Stokes Syndrome.

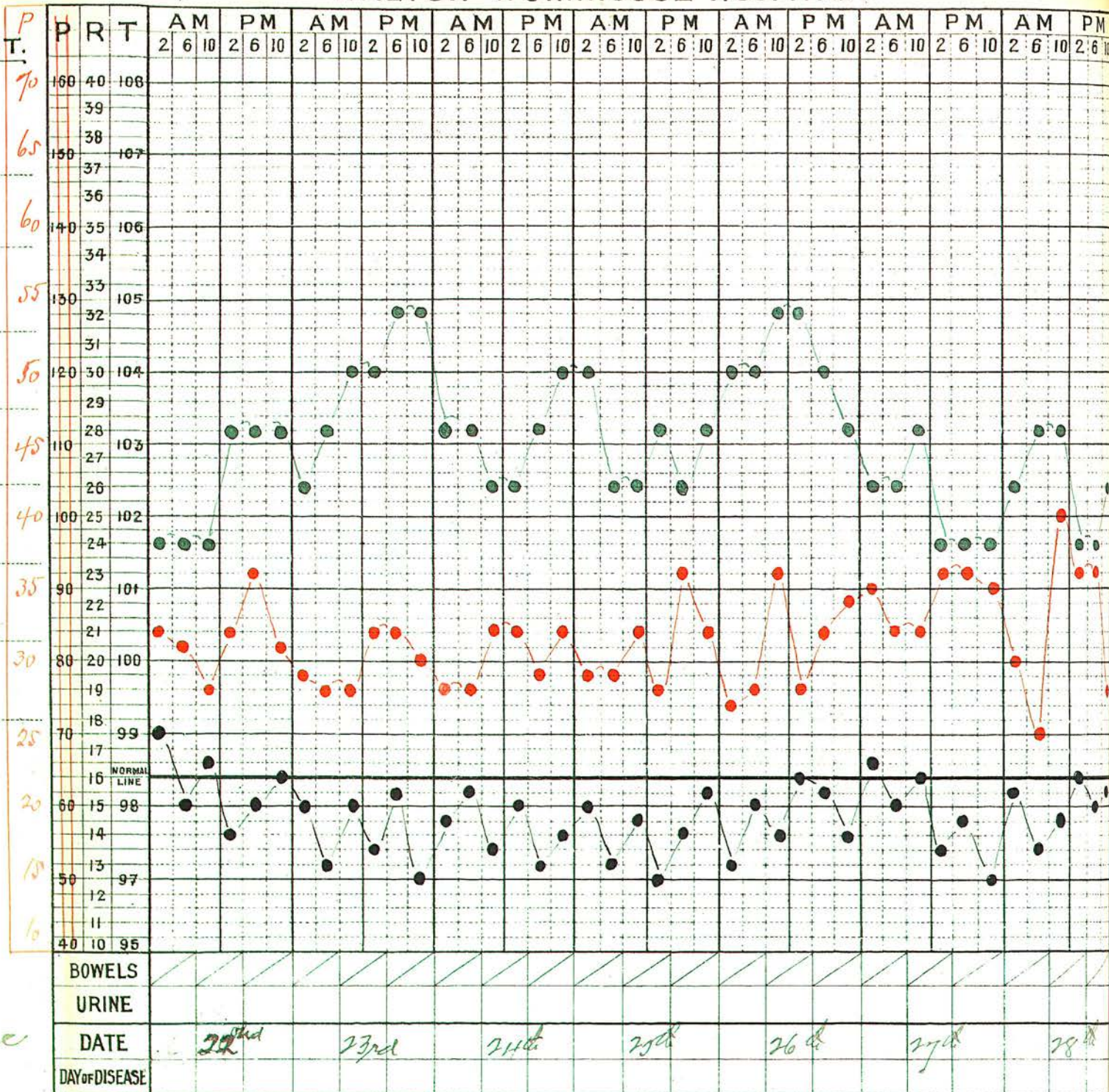
EDGAR GREEN, aet. 64.

Date of admission. June 22nd, 1910.

Complaint. Pain in the limbs and weakness.

General history. From nine years of age his occupation has been that of a painter, both amongst dry and wet paint. His home has always been good. He has taken drink to excess, has suffered from gonorrhœa and has had syphilis. Three years or more ago he commenced to have fits. He would have five or six at a time, and then would be completely free from them for some time. (This important statement will be referred to later in the summary of conclusions). He stated that he would be talking to a person and would suddenly go "right off." These seizures have often caused him to fall in the street. He continued having these "mazy" attacks for two years. After that the attacks came on less frequently and during the last three months he has only suffered from pain in the limbs. For some years

4 HOURS CHART.

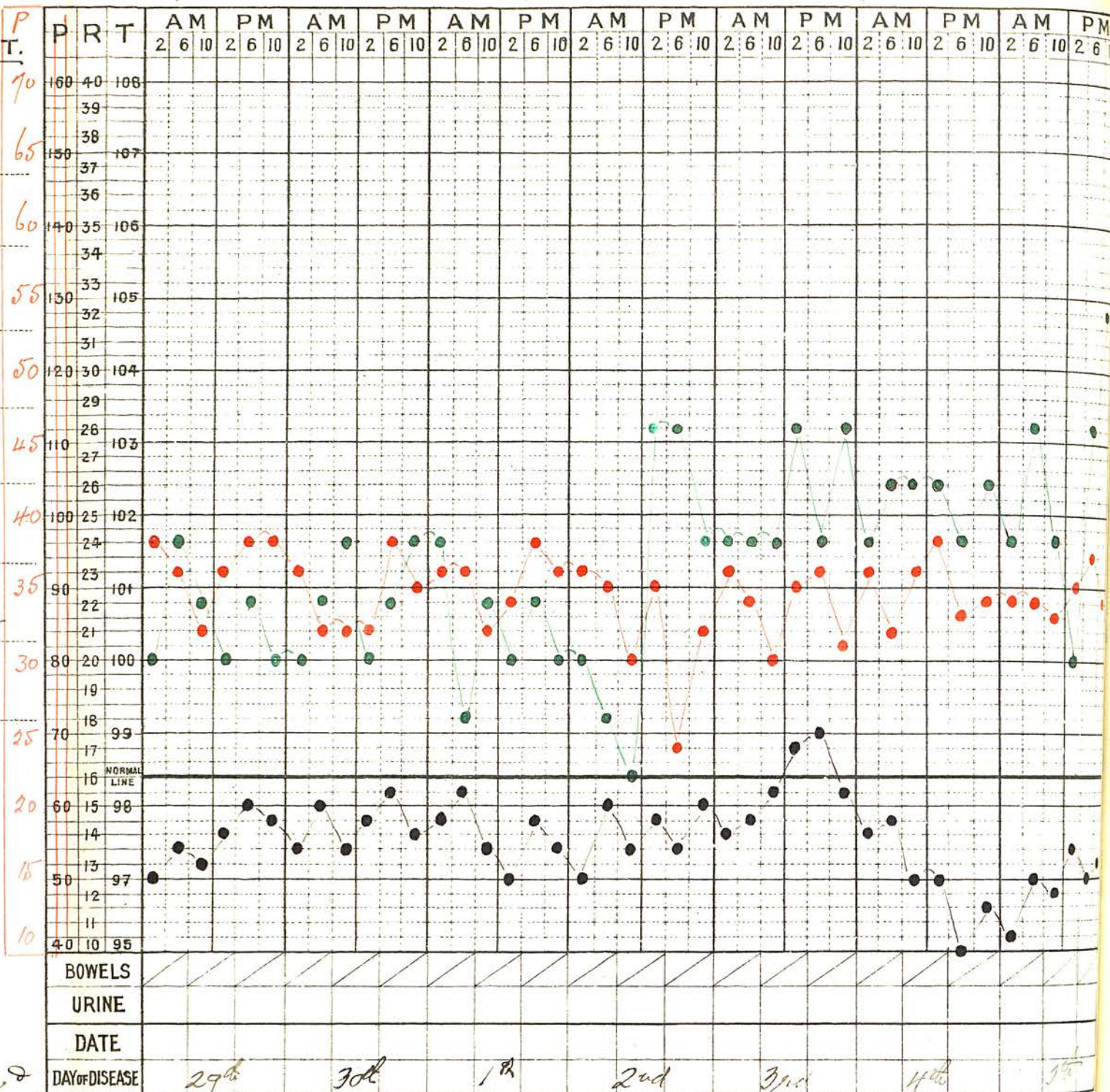
NAME *Edgar
Green*AGE *68 years*OCCUPATION *Painter*ADMISSION *22nd June,
1910.*DISEASE *Heart-block*RESULT *Death
14th July, 1910*

PULSE IN RED

RESPIRATION IN GREEN

TEMPERATURE IN BLACK

4 HOURS CHART.

NAME *Edgar
Green*AGE *68 years*OCCUPATION *Painter*ADMISSION *22nd June,
1910.*DISEASE *Heart block*RESULT *Death
14th July, 1910**June 2
July*

PULSE IN RED

RESPIRATION IN GREEN

TEMPERATURE IN BLACK

WALTON WORKHOUSE HOSPITAL.

4 HOURS CHART.

NAME *Edgar Green*

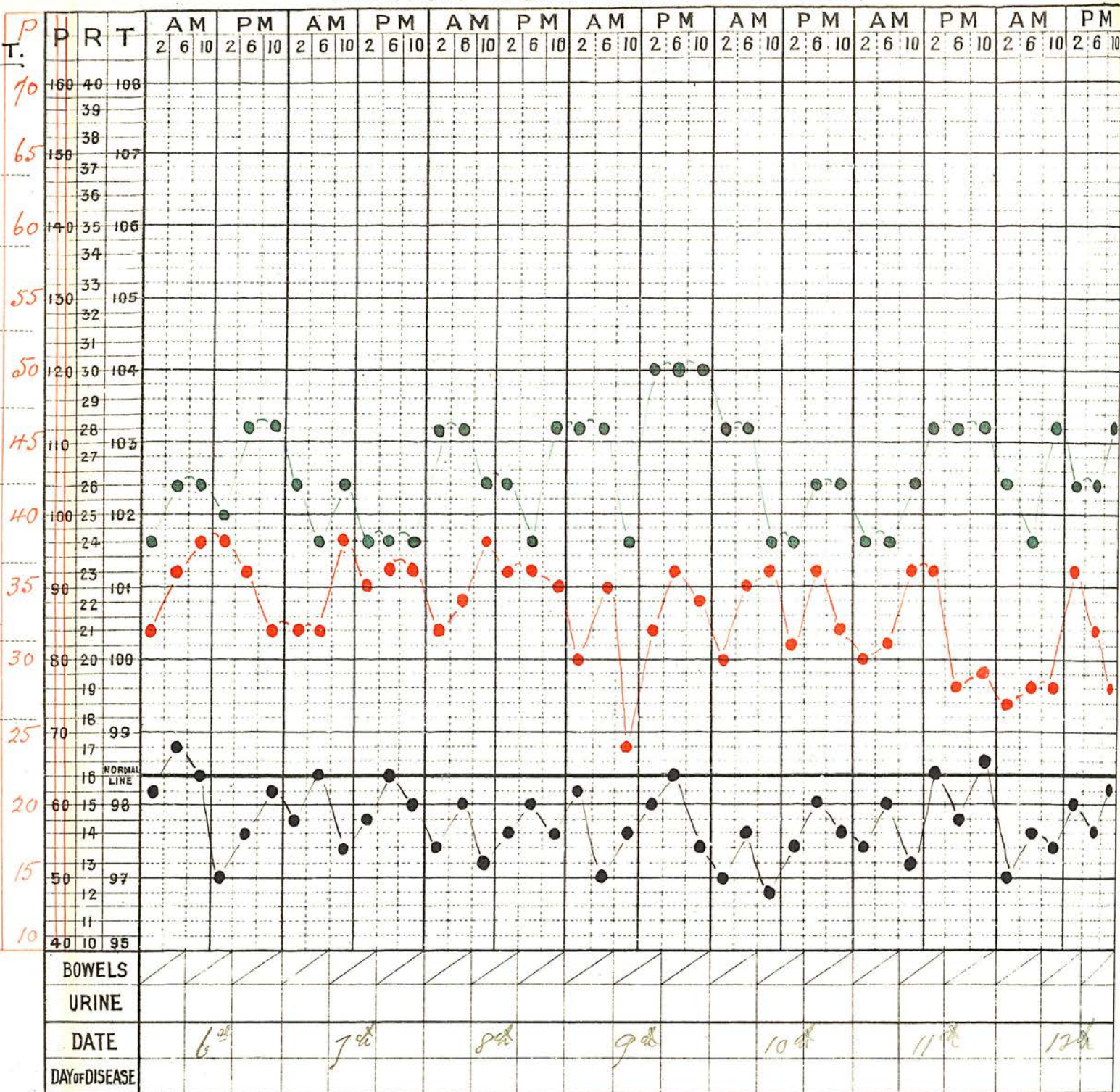
AGE *68 years*

OCCUPATION *Painter*

ADMISSION *22nd June, 1910.*

DISEASE *Heart-block*

RESULT *Death*
14th July, 1910.



PULSE IN RED

RESPIRATION IN GREEN

TEMPERATURE IN BLACK

WALTON WORKHOUSE HOSPITAL.

4 HOURS CHART.

NAME *Edgar
Eisen*

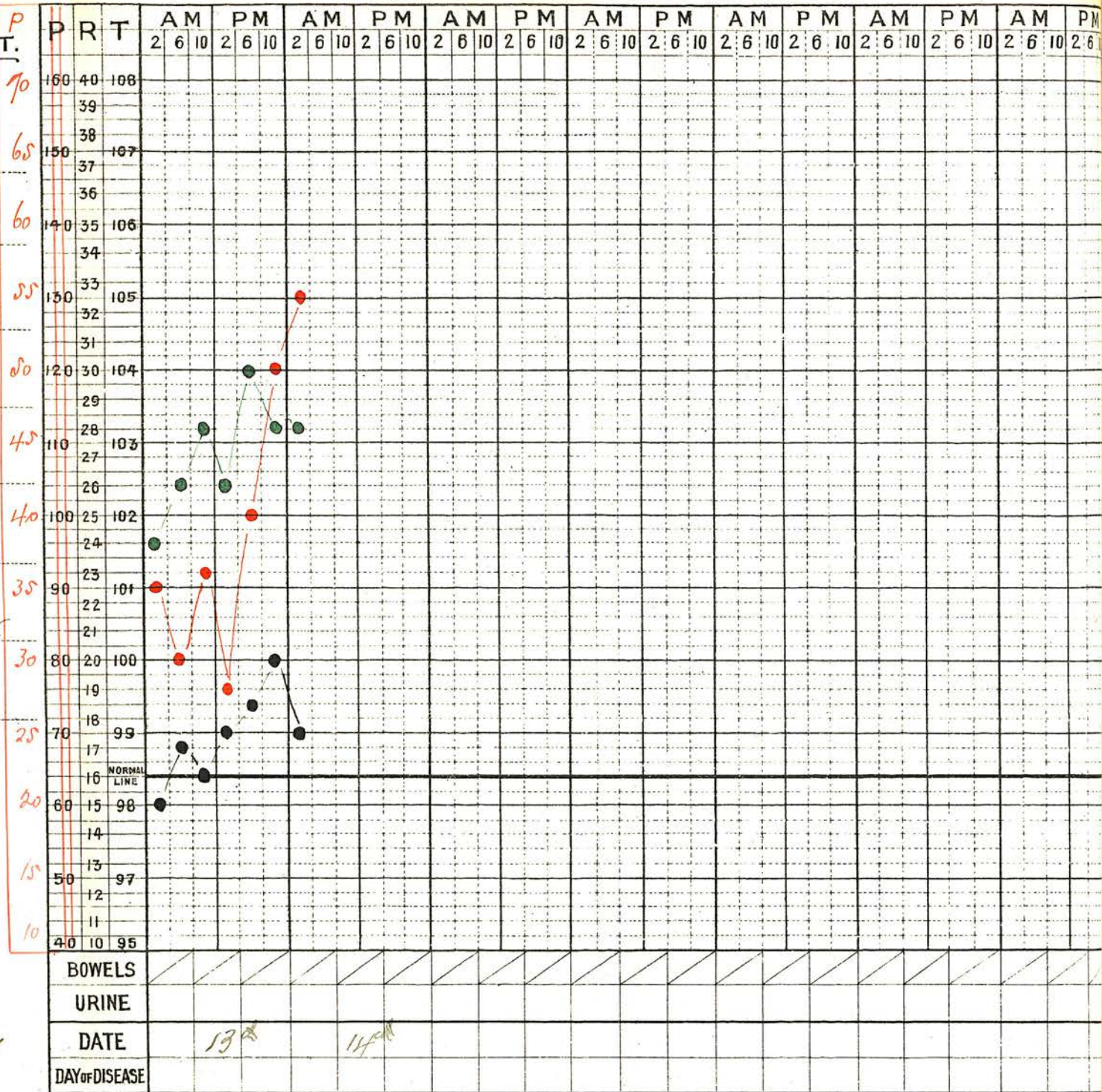
AGE *68 years*

OCCUPATION *Painter*

ADMISSION *22nd June,
1910*

DISEASE *Heart Block*

RESULT *Death
14th July, 1910.*



PULSE IN RED RESPIRATION IN GREEN TEMPERATURE IN BLACK

he has had insufficient food owing to the impossibility of getting work. His relatives say that he has lived a vicious and immoral life.

State on admission He is a fairly well developed man of good intelligence. Finger nails are clubbed. There is slight œdema of the body.

Circulatory system Arteries are hard, thickened and tortuous. The systolic blood pressure is 190mm. of Hg. The pulse rate is 36 and it is perfectly regular in force and frequency (vide pulse chart). Pulsations are visible in the veins of the neck, more marked on the left side. The heart extends two inches to the right and four-and-a-half inches to the left of the middle line. On auscultation in the mitral area there is a faint presystolic and a loud blowing systolic murmur which is propagated all over the chest. There is a slight tricuspid regurgitation. The second sound is closed.

Nervous system. There is nothing of interest to note.

Alimentary system The liver is enlarged.

Renal system. Urine sp. gr. 1012. The reaction is neutral. Urates are present. There is no albumen.

Progress and treatment of the case. Patient was treated with a mixture containing potassium iodide grs. xv., and sodium nitrite grs. ii. for a fortnight. During this fortnight hypodermic injections of strychnine were given in doses of $\frac{1}{60}$ of a grain three times daily. His condition remained the same. The pulse was still about 36 in rate and was regular. Atropine was then tried in doses of $\frac{1}{100}$ of a grain four-hourly for a few days. This did not have any effect on the pulse rate or on the frequency of the auricular systole. Vagus stimulations (pressure) were tried without any result. Whilst in hospital his appetite was very bad, and he scarcely ate any food. He became much weaker and strychnine gr. $\frac{1}{100}$ was administered four-hourly. He died on the 14th July without showing any symptom of note. He had no seizures or syncopal attacks whilst in hospital.

Graphic records. Tracings demonstrated complete auriculo-ventricular heart-block.

Auricular frequency 67·05 per minute.

Ventricular frequency 35·4 per minute

A diagrammatic interpretation of the events recorded is placed below tracing 1, and shows complete dissociation of the rhythms of the auricles and ventricles. The downstroke on the venous tracing indicates the commencing point of the analysis, and No. 1, 2, 3 etc. indicate ventricular systoles.

The other tracings show similar phenomena, the block always remaining complete.

Pathological Examination of the Heart.

- Heart. The heart weighs 330 grammes.
- External measurements. Circumferential measurement in the long axis of the heart, 35cm.; circumferential measurement around the auriculo-ventricular groove, 27cm.; length, 13.9cm.; breadth, 9cm.; antero-posteriorly, 8.5cm.
- Long diameters of the cavities. Right auricle, 5.8cm.; right ventricle, 6.1cm.; left auricle, 5cm.; left ventricle, 7.7cm.
- Epicardium. The epicardium is thick, especially over the right auricle and right ventricle, where there are large, thick, "milk-spots."
- Right auricle. The endocardium is slightly thickened. The muscle is not hypertrophied generally, and the tænia terminalis and the pectinate muscles are not specially thick. No change can be seen in the wall of the junction of the superior vena cava and auricle. The foramen ovale is closed.
- Projecting upon the right septal wall, below the lower side of the pars membranacea septi and immediately above the anterior end of the line of attachment of the septal cusp of the tricuspid valve, there is a firm, calcareous subendocardial nodule, 8mm. in diameter. The greater part of this nodule lies above the line of attachment of the septal cusp of the tricuspid valve, but the lower part of the nodule lies underneath the basal part of the anterior end of the cusp. This nodule constitutes the posterior end of the calcareous, horse-shoe shaped ring, which will be described later.
- Tricuspid orifice. The tricuspid orifice is 10.5cm. in circumference. The valve segments are not thickened, and the chordæ tendineæ are neither shrunk nor thickened.
- Right ventricle. The musculature is not hypertrophied, and no fibrous patches can be seen either in the wall of the cavity or in the papillary muscles.
- Pulmonary orifice. The pulmonary orifice is 7cm. in circumference. The pulmonary valve and artery are healthy.
- Left auricle. The endocardium is only slightly thickened. The musculature is moderately hypertrophied.
- Mitral orifice. The mitral orifice is 8cm. in circumference. The basal portion of both cusps are both thick and calcareous, but the apical portions are not thickened, and the chordæ tendineæ are not thick or shrunk. The relation of the mitral cusps to the calcareous ring will be described later.

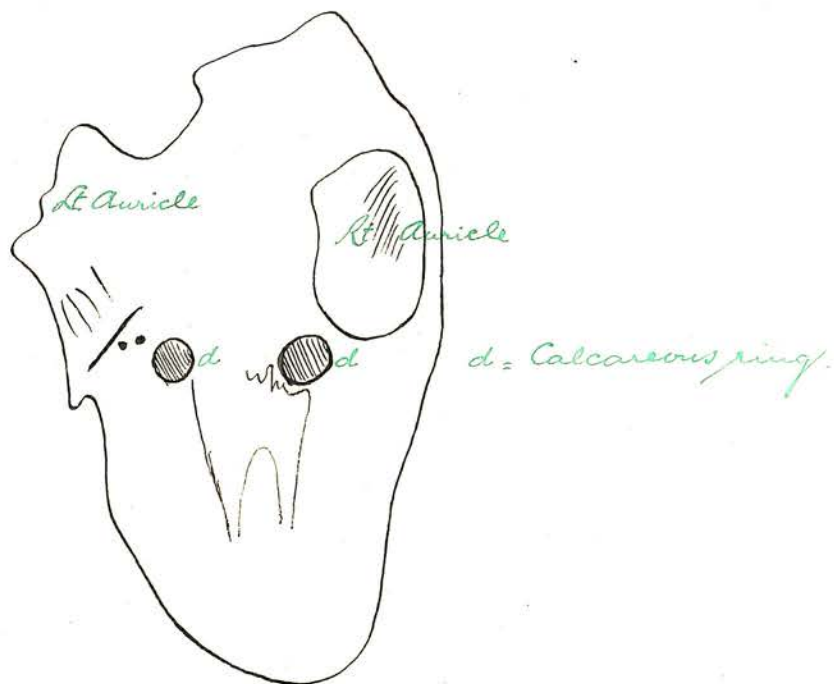
EDGAR GREEN



G 1. Left Ventricle, Aortic Cusp of Mitral Valve, left Auricle and Appendix and, darkly printed, the cavity of the right Auricle. The calcareous mass is seen in section and the thick Coronary vessels are seen.

Edgar Green

Diagram of photograph No. 1



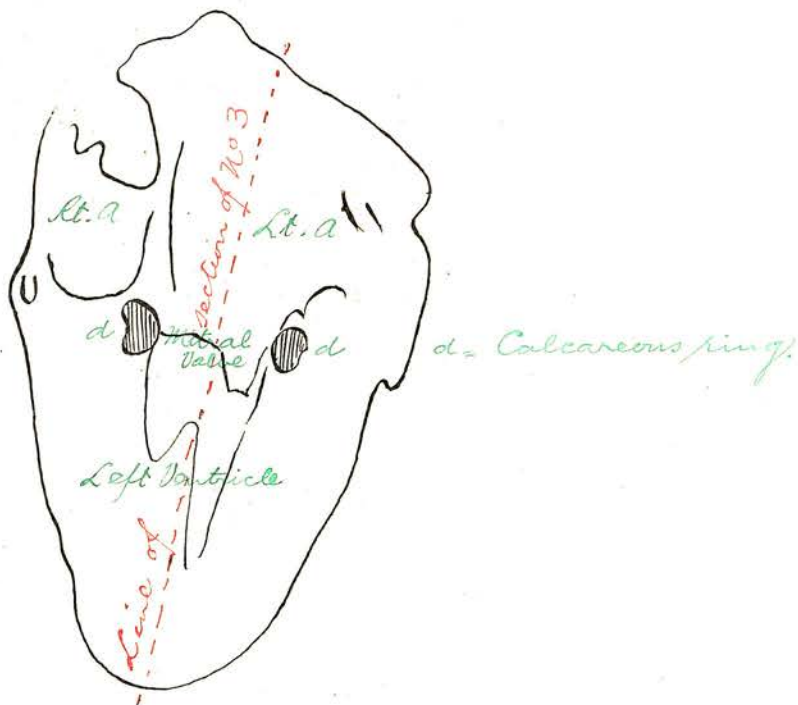
EDGAR GREEN



G 2. Outer half of left Ventricle with Mitral Valve (posterior segment), left Auricle and Appendix, and portion of right Auricle with Coronary Sinus. This view shows the calcareous ring around the Mitral Orifice.

Edgar Green

Diagram of photograph N^o 2,
showing line of section of N^o 3.



EDGAR GREEN



G. 3. View of section through the middle of the posterior segment of the Mitral Valve, showing the large white calcareous mass under the valve segment, yet involving it from below.

4. To show transverse sections of the calcareous ring in relation to the aortic valve. The ring lies immediately below the deepest parts of the anterior and right posterior sinuses of valsalva.



G4

1 and 2 show the parts exposed by a longitudinal section through the mid point of line of attachment of the anterior cusp of the aortic valve. In 3 the line of section passes through the corresponding point of the right posterior cusp.

1. Aorta
2. Half of anterior cusp of aortic valve.
3. Anterior sinus of valsalva
4. Orifice of right coronary artery
5. Left auricular appendix
6. Aortic cusp of mitral valve
7. Pulmonary artery
8. Right posterior cusp of aortic valve
9. Right posterior sinus of valsalva
10. Conus arteriosus
11. Left ventricle

Edgar Green



G5



G6

- G6. 1. Anterior cusp of Aortic valve
2. Right posterior cusp of aortic valve
3. Aortic (anterior) cusp of mitral valve



G7

- G7. 1. Aortic cusp of mitral valve
2. Auricular Septum
3. Below the calcareous ring in the Ventricular septum
4. Septal cusp of tricuspid valve

- G5. 1. Superior vena cava. 2. Annulus ovalis
- 3, 5. The calcareous projection above anterior end of line of attachment of and below basal part of and anterior end of, septal cusp of tricuspid valve.
4. Septal cusp of tricuspid valve. 5. Infundibular cusp.
6. Wall of conus arteriosus. 7. Aorta

Edgar Green
Posterior

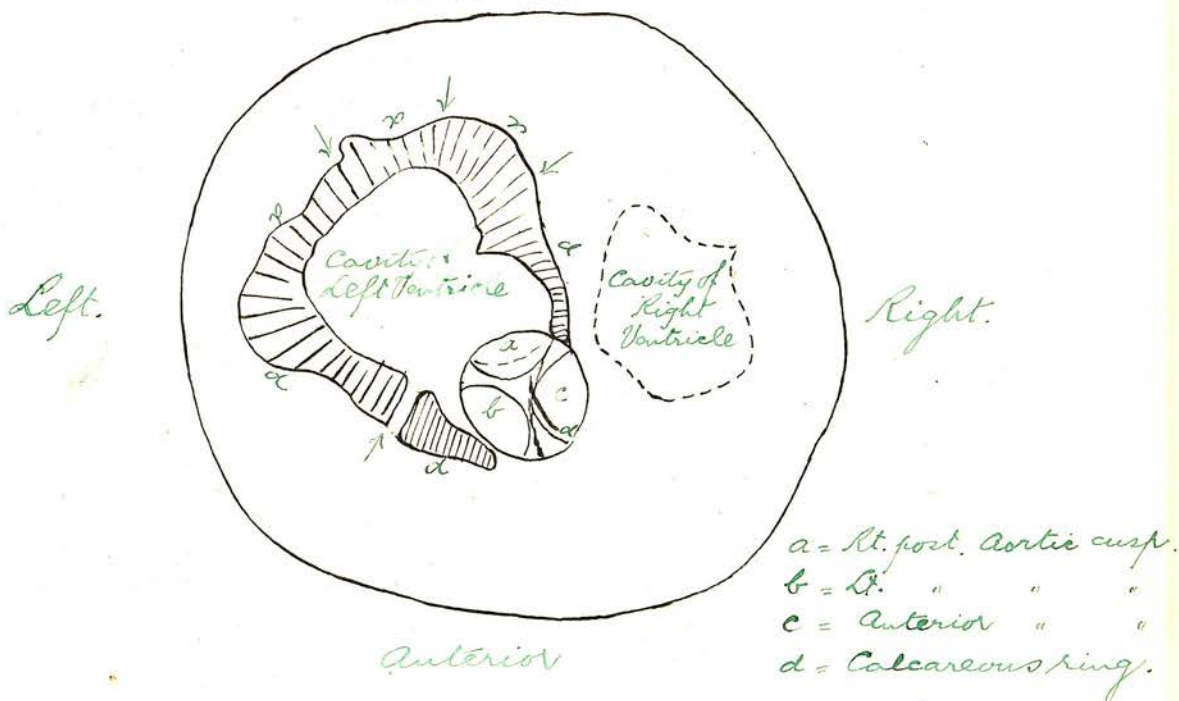


Anterior
X-ray photograph showing the
Calcareous ring

Edgar Green

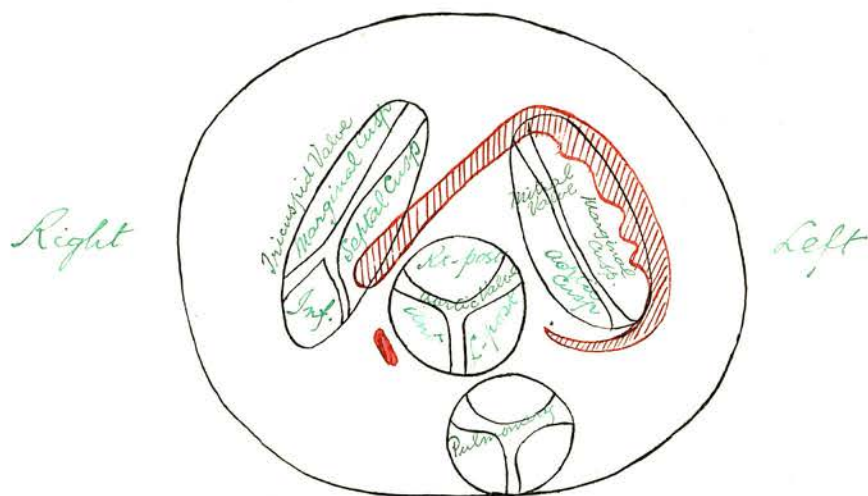
Diagram of the
X-ray photograph

Posterior



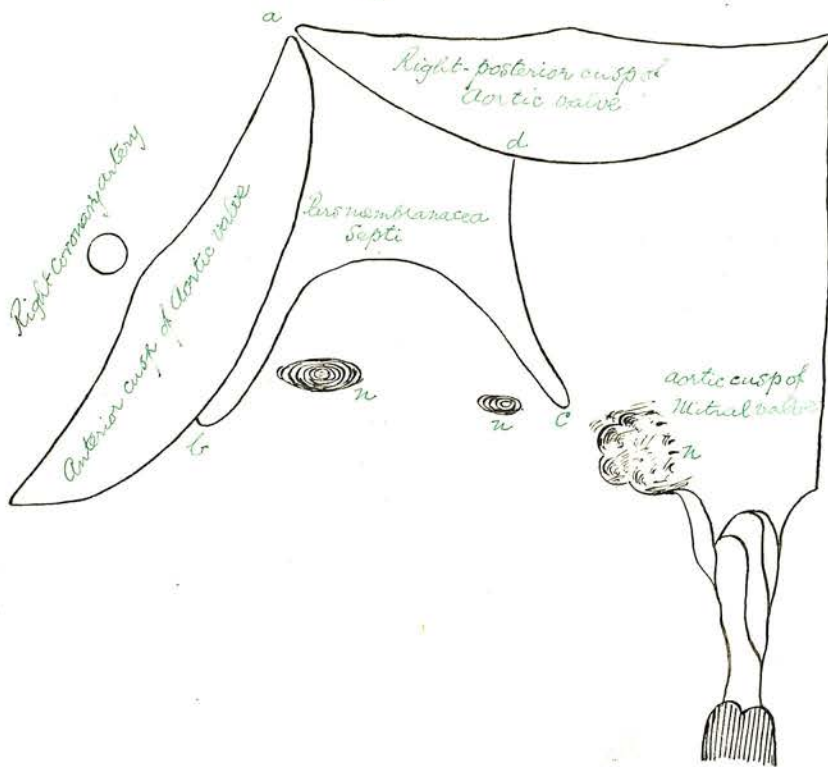
The heart had been considerably cut up,
and was pieced together later for the
purpose of taking the X-ray photograph,
so the ring is cut transversely in these
places — ↓.

Edgar Green



The Calcareous ring in red

Eagan Green



$a-b = 16 \text{ mm.}$
 $b-c = 16 \text{ mm.}$
 $c-d = 10 \text{ mm.}$
 $d-a = 8 \text{ mm.}$

$n = \text{calcareous nodule}$

Edgar Green



To show the relation of the calcareous, posterior,
and of the shoe shaped ring to the pars
membranacea septi.

Left ventricle. The muscle wall is moderately hypertrophied, being 1.5cm. thick near the base, and 1.8cm. thick at the apex of the cavity. Except for the calcareous ring in the basal part of the ventricle the muscle appears to be healthy.

Aortic orifice. The aortic orifice is 6.5 cm. in circumference. The cusps are slightly thickened, but not shrunk. There is a calcareous plate in the wall of the anterior sinus of Valsalva. The wall of the ascending aorta presents nodular patches of atheroma, and in the walls of the coronary arteries and their branches there are also atheromatous patches.

Calcareous ring. In the basal part of the ventricular musculature there is a large, horse-shoe shaped, calcareous ring, lying in the plane of the mitral orifice. Anteriorly the ring commences just below the deepest part of the left-posterior cusp of the aortic valve, yet not involving the cusp. From this point onwards to the outer end of the aortic cusp of the mitral valve, the ring projects upon the endocardial surface of the left ventricle as a prominent, irregular and nodular ridge. The ring passes close under, but does not involve this cusp. On transverse section at the outer end of the line of attachment of the aortic cusp of the mitral valve, the ring is ovoid and measures 7mm. by 6mm. Thence the ring passes around in the ventricular musculature close under, and in places even involving, the marginal cusp of the mitral valve, for the ring spreads into this cusp from its ventricular surface, and bulges the cusp upwards towards the auricle. At the mid-point of the line of attachment of the marginal cusp the ring is thicker than elsewhere, for here, it is on transverse section of irregular quadrilateral form, and measures 8mm. by 6mm. (see photograph).

Passing under the inner ends of the lines of attachment of the mitral cusps, the ring forms a ridge projecting upon the endocardial surface. Thence the ring passes forwards through the septum obliquely from left to right, passing 5mm. below the anterior point of the mouth of the coronary sinus. Viewed from the left side the posterior end of the ring is represented by a dense, greyish-white opaque area, with a convex upper margin, (see diagram), which projects into the lower part of the pars membranacea septi below the point of junction of the anterior and right-posterior cusps of the aortic valve. The pars membranacea septi is thus seen to be encroached upon from below by the calcareous ring. Viewed from the right side of the septum, the ring is seen to terminate as the calcareous nodular projection already described, above the anterior end of the line of attachment, and underneath the anterior end of the basal part of the septal cusp of the tricuspid valve.

WALTON WORKHOUSE HOSPITAL.

4 HOURS CHART.

NAME *Thomas
Gates*

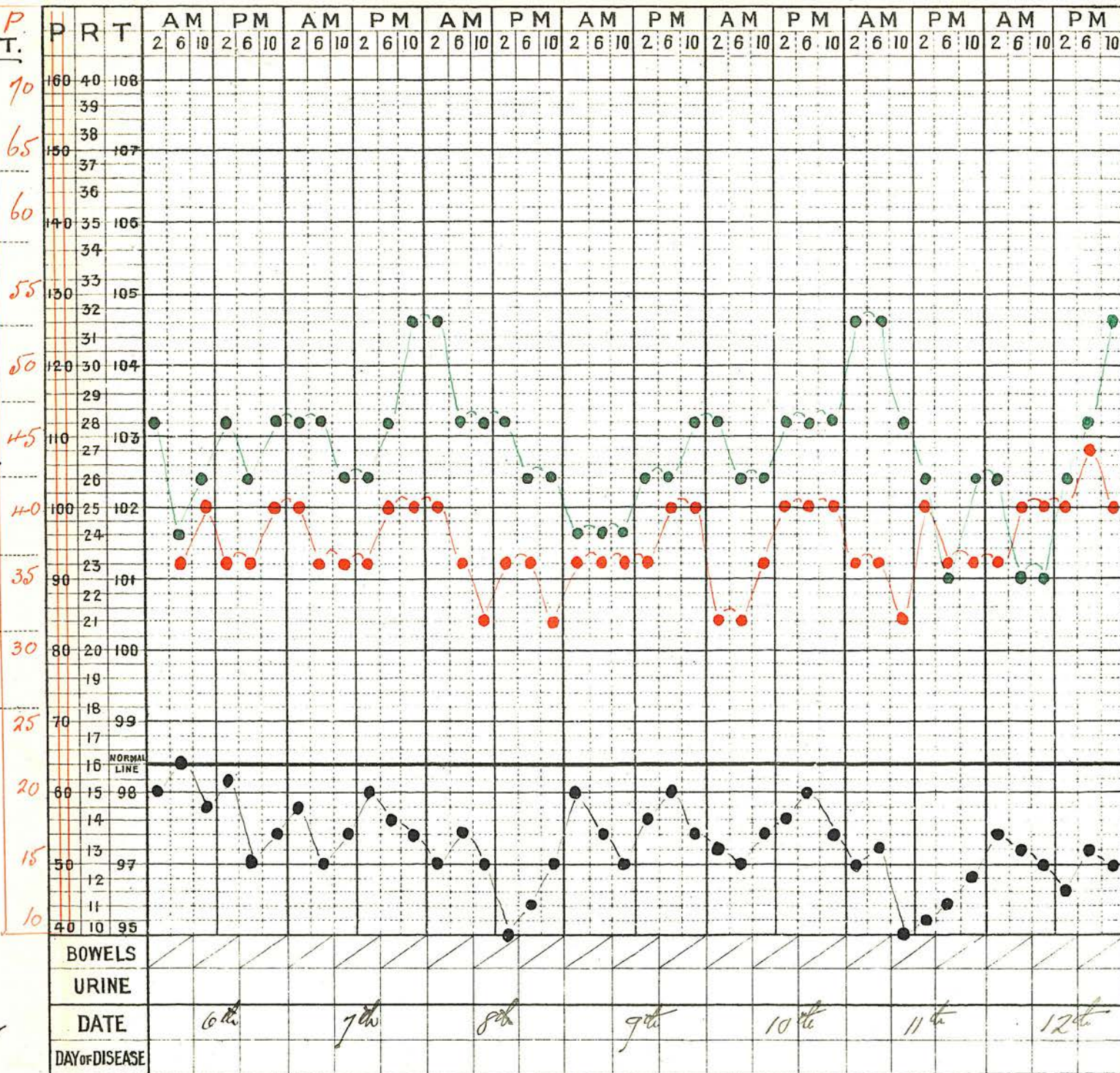
AGE *62 years*

OCCUPATION *Labourer*

ADMISSION *6th July,
1910*

DISEASE *Heart
block*

RESULT *Death
16th July, 1910*



July

PULSE IN RED

RESPIRATION IN GREEN

TEMPERATURE IN BLACK

4 HOURS CHART.

NAME _____

Thomas
Gates

AGE

62 years

OCCUPATION

Labourer
6th July
1910

ADMISSION

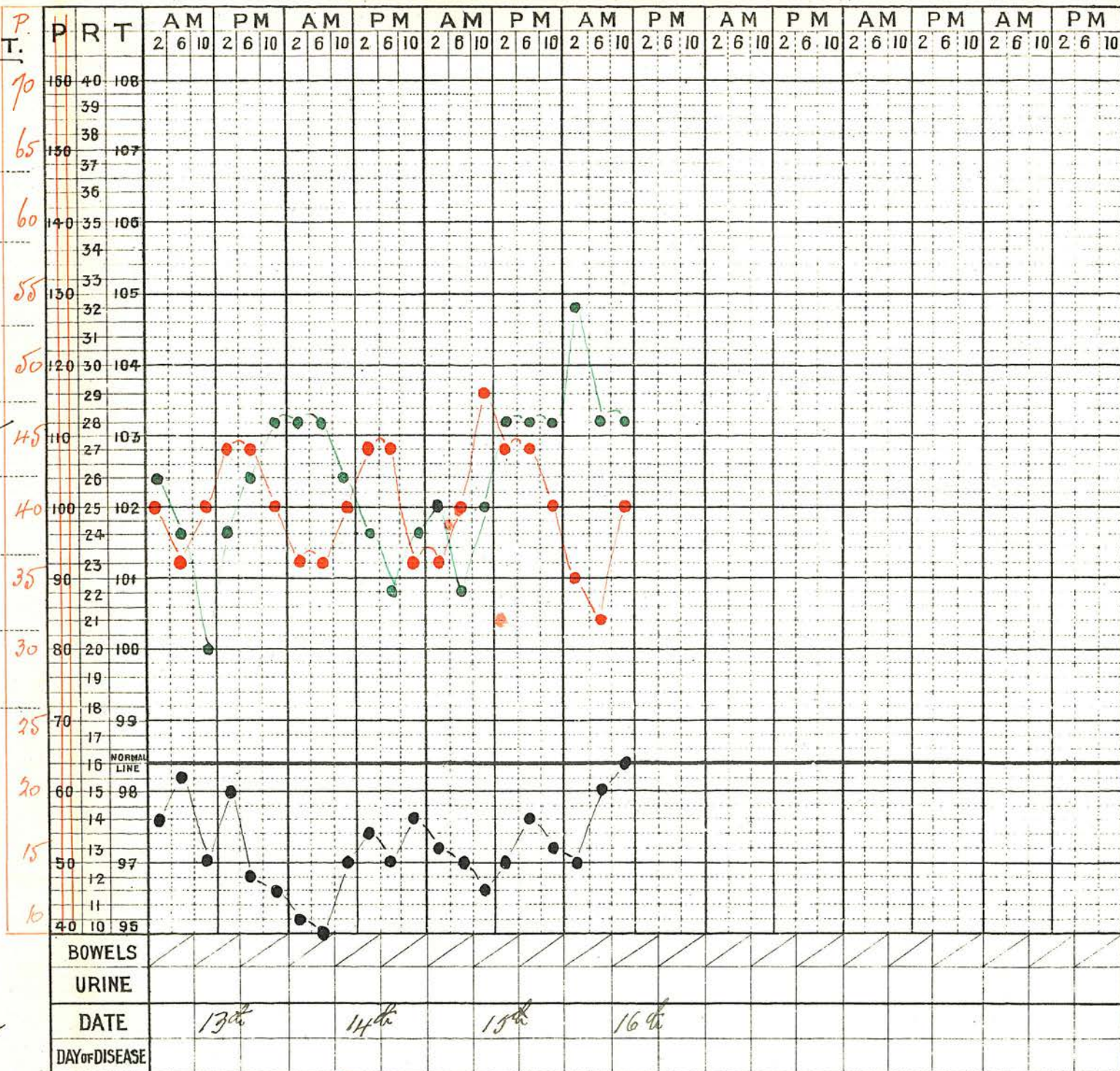
6th July,
1910

DISEASE

Heart
block

RESULT

RESULT Death
16th July, 1910.



July

PULSE IN RED

RESPIRATION IN GREEN

TEMPERATURE IN BLACK

CASE 3.

Complete Heart-Block with History of Adams-Stokes Syndrome.

THOMAS YATES, aet. 62.

Date of admission. July 6th, 1910.

Complaint. Giddiness and cough.

General history. His occupation is that of a machinist in a ropery works, and his work has always been strenuous. He is a married man with two of a family. He has suffered from rheumatism, has not had syphilis and has always been a temperate man. His illness began three months ago with "whizzing and giddiness in the head." He was afraid to eat anything, as swallowing often started a "mazy bout," and he also noticed that an attack of coughing was often followed by a fainting attack. He described an attack he had before admission in the following terms:—"A weakness came over me, and then all the good went out of my legs, my head and forehead got as hot as fire, and then I lost consciousness for a little. When I came back I was lying on the floor and felt sick."

Since admission he has had transient attacks of giddiness, but has not lost consciousness and has had no convulsive seizures.

State on admission. His respirations and temperature are normal and the pulse is 36 in rate. His face is cyanosed and his breathing is rather laboured.

Circulatory system. Pulsations are visible in the jugular veins. The apex beat is in the fifth interspace just outside the nipple line. The right border of the heart is 2 inches to the right, and the left is 5 inches to the left of mid-sternum. On auscultation there is a soft blowing mitral systolic murmur.

The arteries are thickened. The pulse is regular and varies in rate between 36 and 40 (*vide* pulse chart). The systolic blood pressure is 230mm. of Hg.

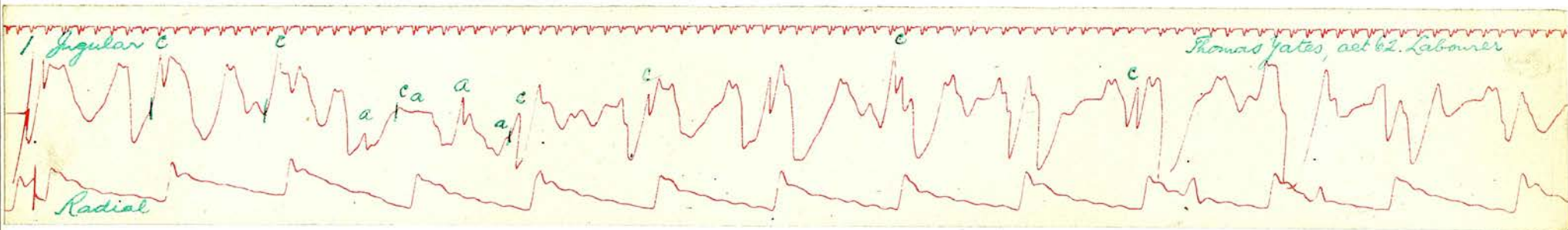
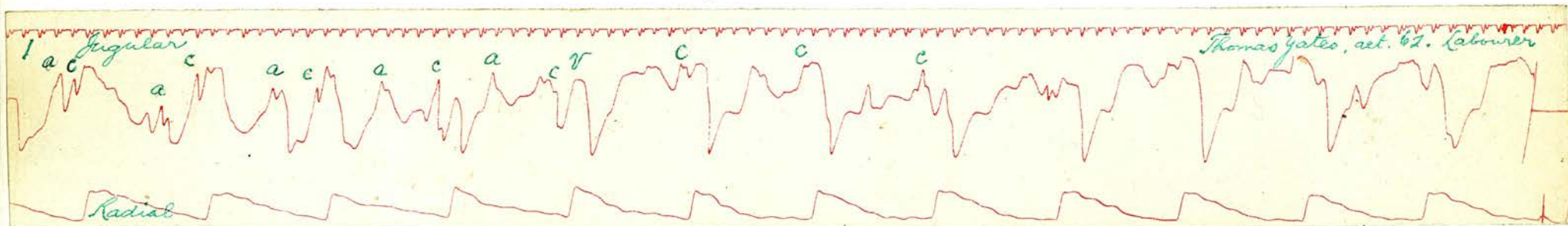
Respiratory system. The chest is large and barrel-shaped, and the expectoration is profuse and frothy.

Nervous system. Excepting the giddiness there is nothing of interest to note.

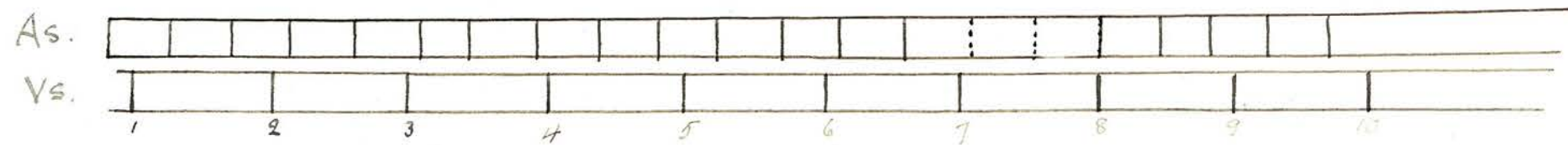
Renal system. Urine: Sp. gr. is 1012. The reaction is acid, and there is a slight trace of albumen.

Progress and treatment of the case. Patient has had slight attacks of giddiness, but has not lost consciousness. Some days after admission patient's heart began to fail and his urine was scanty. Hypodermic injections of strychnine gr. $\frac{1}{30}$ were administered every six hours. Brandy was also administered, and diffusible stimulants. He died on the 16th of July, 1910.

Thomas Gates



Thomas Gates



As = Auricular Systole

Vs = Ventricular Systole

∴ = Probable auricular Systole

Complete Heart-block

Graphic records. Tracings show complete auriculo-ventricular heart-block.

Auricular frequency 67·5 per minute.

Ventricular frequency 37·5 per minute.

The schematic diagram of tracing No. 1, constructed as in the preceding case, shows that the auricular and ventricular rhythms are wholly independent of each other.

The other tracings show a similar condition of affairs.

Pathological Examination of the Heart.

Heart. The heart weighs 525 grammes.

External measurements. Circumferential measurement in the long axis, 39cm.; circumferential measurement around the Auriculo-ventricular groove, 30·7cm.; length, 15·5cm.; breadth, 10·5cm.; antero-posteriorly, 8·1cm.

Epicardium. The epicardium is thick, especially over the auricles and the apical portion of the left ventricle. There is a thick layer of adipose tissue over the conus arteriosus.

Right auricle. The endocardium is considerably thickened. The foramen ovale is closed. There is a well-developed, fenestrated valve of Eustachius, shaped somewhat like the aortic cusp of the mitral valve. Its attached margin is 2·3cm. in length, and from the mid-point of the line of attachment to the apex of the valve measures 2·3cm. The auricular muscle is markedly hypertrophied. The tænia terminalis is 1cm. in diameter, and while the pectinate muscles are mostly 3mm. in diameter, others are as much as 5mm. in diameter. No pathological change can be detected in the region of the sino-auricular node. The pars membranacea septi is thin and translucent.

Tricuspid orifice. The tricuspid orifice is 11·5cm. in circumference. The valve segments and the chordæ tendineæ are healthy.

Right ventricle. The endocardium presents numerous small patches of fibrous thickening. The musculature is hypertrophied. The thickness of the muscle wall posteriorly is 6mm.

Pulmonary orifice. The pulmonary orifice is 7·3cm. in circumference. The valve and artery are healthy.

Left auricle. The endocardium is slightly thickened and the musculature is hypertrophied.

Mitral orifice. The mitral orifice is 8·4cm. in diameter. The valve segments present slight, diffuse thickening, and small, yellowish fibrous areas in their

Y1. Right Heart

1. Superior vena cava
2. Tricuspid valve
3. Valve of tricuspid valve
4. " " Tricuspid valve
5. Mouth of Coronary Sinus
6. Marginal cusp of Tricuspid Valve
7. Septal cusp of Tricuspid Valve
8. Infundibular cusp of Tricuspid Valve
9. Wall of conus arteriosus
10. Pulmonary Artery
11. Aorta

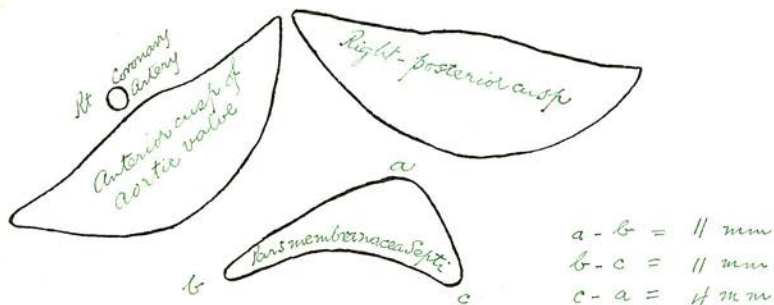
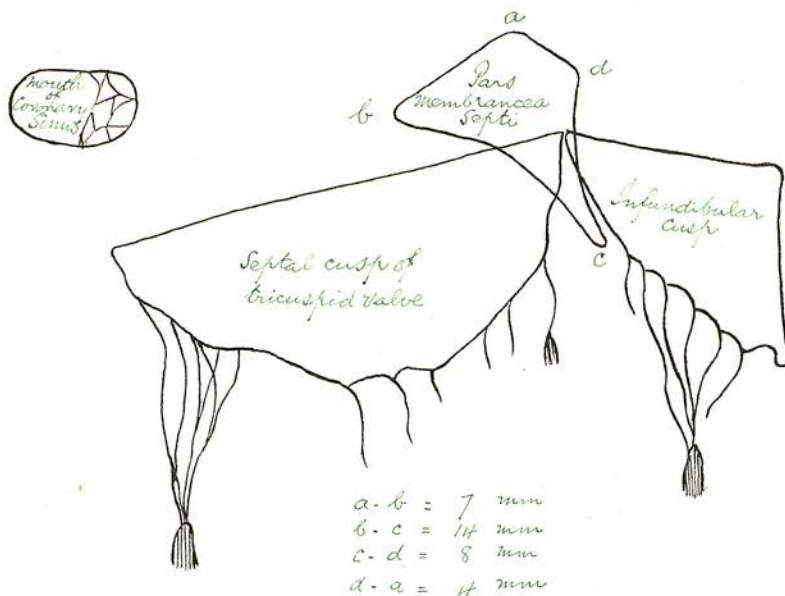


Y2. Left Heart



Thomas Yates

Thomas Gates



Two diagrams to shew the relation of the pars membranacea septi to the tricuspid and aortic valves.

substance, but they are not calcareous. The chordæ tendineæ are slightly thick but not shrunken. The apices of the papillary muscles are fibrous.

Left ventricle. The endocardium shows a few patches of fibrous thickening. The muscle is not fibrous, but is hypertrophied. The muscle wall is 1.8cm. thick near the base of the cavity, and 1.3cm. thick at the apex.

The pars membranacea septi is a small translucent triangular area—the sides of which measure 11mm. 11mm. and 4mm. respectively—below the right posterior cusp and the junction of this cusp with the anterior cusp on the aortic valve.

Aortic orifice. The aortic orifice is 7.5cm. in circumference. The anterior and left-posterior cusps are slightly thickened. The central portion of the right posterior cusp is calcareous, but the calcification does not extend into the line of attachment of the cusp nor into the musculature adjacent thereto. The endocardium in the deepest portion of all the sinuses of valsalva is thick and fibrous.

The wall of the ascending aorta presents a few nodular patches of atheroma. The walls of the coronary arteries contain many small atheromatous nodules, but the vessels are not calcareous.

CASE 4.

Heart-Block with Adams-Stokes Syndrome.

MARY RILEY, Aet. ?.

Date of admission. June 24th, 1910.

Complaint. Weakness and "mazines." Cough.

General history. Patient has always had to do a lot of hard work. She has had no serious illnesses. For the last year she has felt "mazy" if she undergoes any exertion, and this feeling has become much worse within the last ten weeks, the "mazy" feeling progressing to a fainting attack. She often has pain over the region of the heart but this pain is not associated with an attack. During the present illness her memory has become impaired. If she tries to clean or to do anything she has a fainting attack. There is no history of syphilis and she is a temperate woman.

Since admission she has had a number of syncopal attacks, but so far has not been observed in one. She states that she has had attacks during the night and feels as if she were going to die when they come on.

She says that when an attack comes on she feels "mazy" and a far-a-way feeling comes over her, then she loses consciousness and cannot recollect where she is or what has happened when consciousness returns.

WALTON WORKHOUSE HOSPITAL.

4 HOURS CHART.

NAME

*Mary
Riley*

AGE

1

OCCUPATION

*House-
wife*

ADMISSION

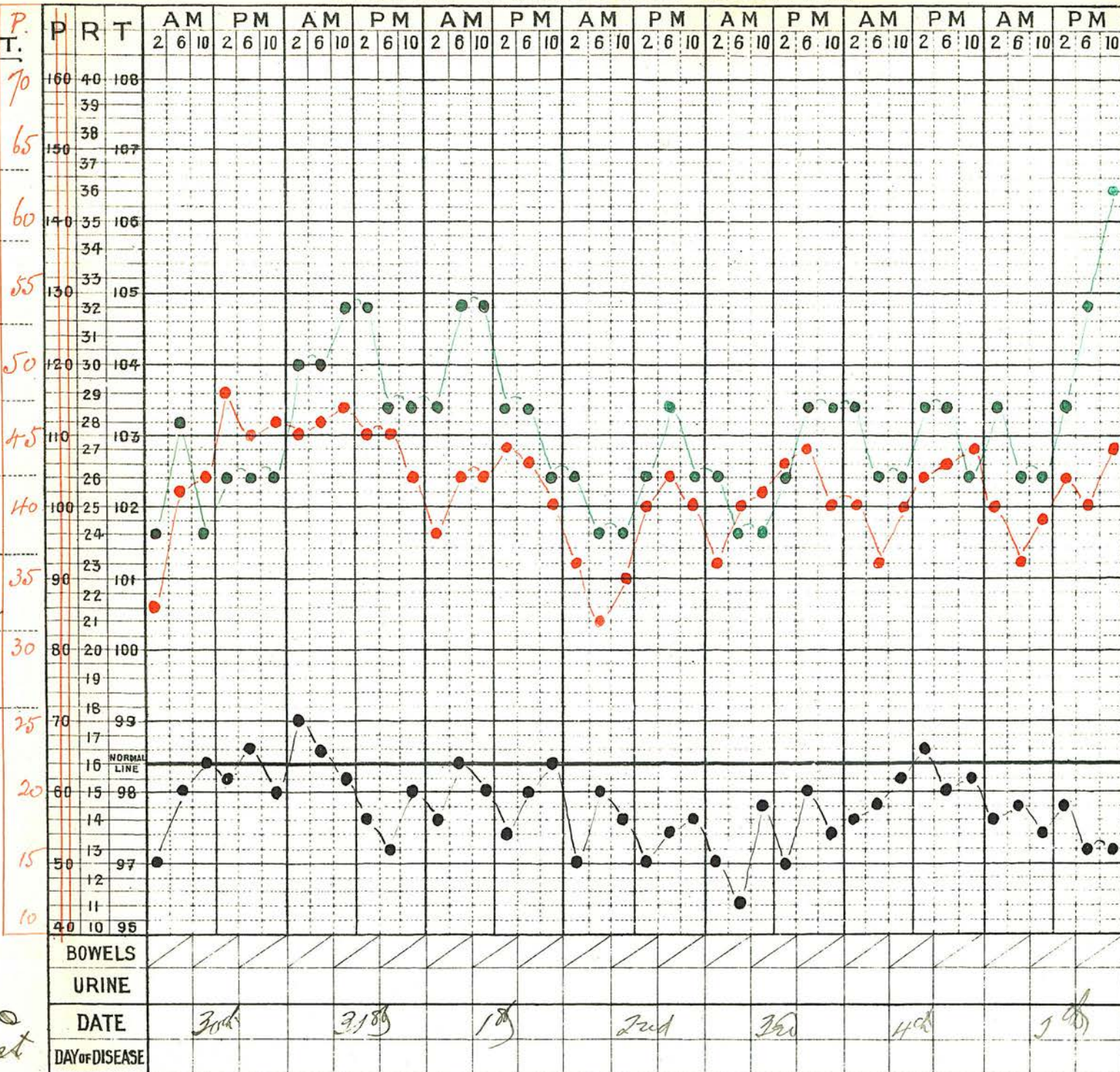
*29th June,
1910*

DISEASE

Heart block

RESULT

*Death
24th July, 1910.*



PULSE IN RED

RESPIRATION IN GREEN

TEMPERATURE IN BLACK

June 8 *July* *August*

No. 2.

WALTON WORKHOUSE HOSPITAL.

4 HOURS CHART.

NAME

Mary
Kilby

AGE

?

OCCUPATION

House-
wife

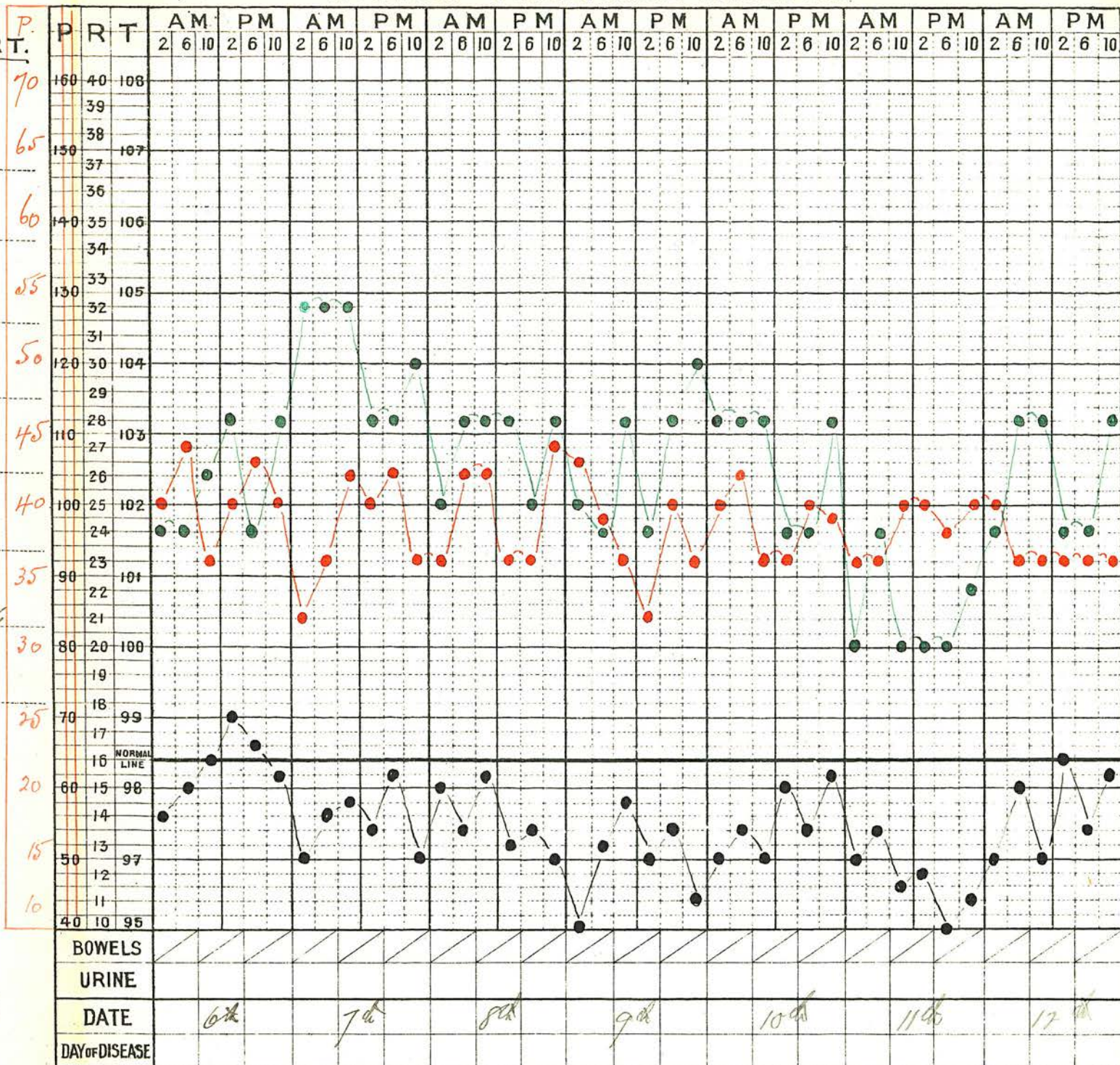
ADMISSION

29th June,
1910

DISEASE

Heart block

RESULT

Death
24th July, 1910.

PULSE IN RED

RESPIRATION IN GREEN

TEMPERATURE IN BLACK

AGE

OCCUPATION Housewife

ADMISSION 29 June
1910

DISEASE Heart block

RESULT Death
24th July, 1910.

BOWELS

URINE

DATE _____

DAY OF DISEASE

PULSE IN RED

RESPIRATION IN GREEN

TEMPERATURE IN BLACK

WALTON WORKHOUSE HOSPITAL.

4 HOURS CHART.

NAME

Mary Riley

AGE

?

OCCUPATION

Housewife

ADMISSION

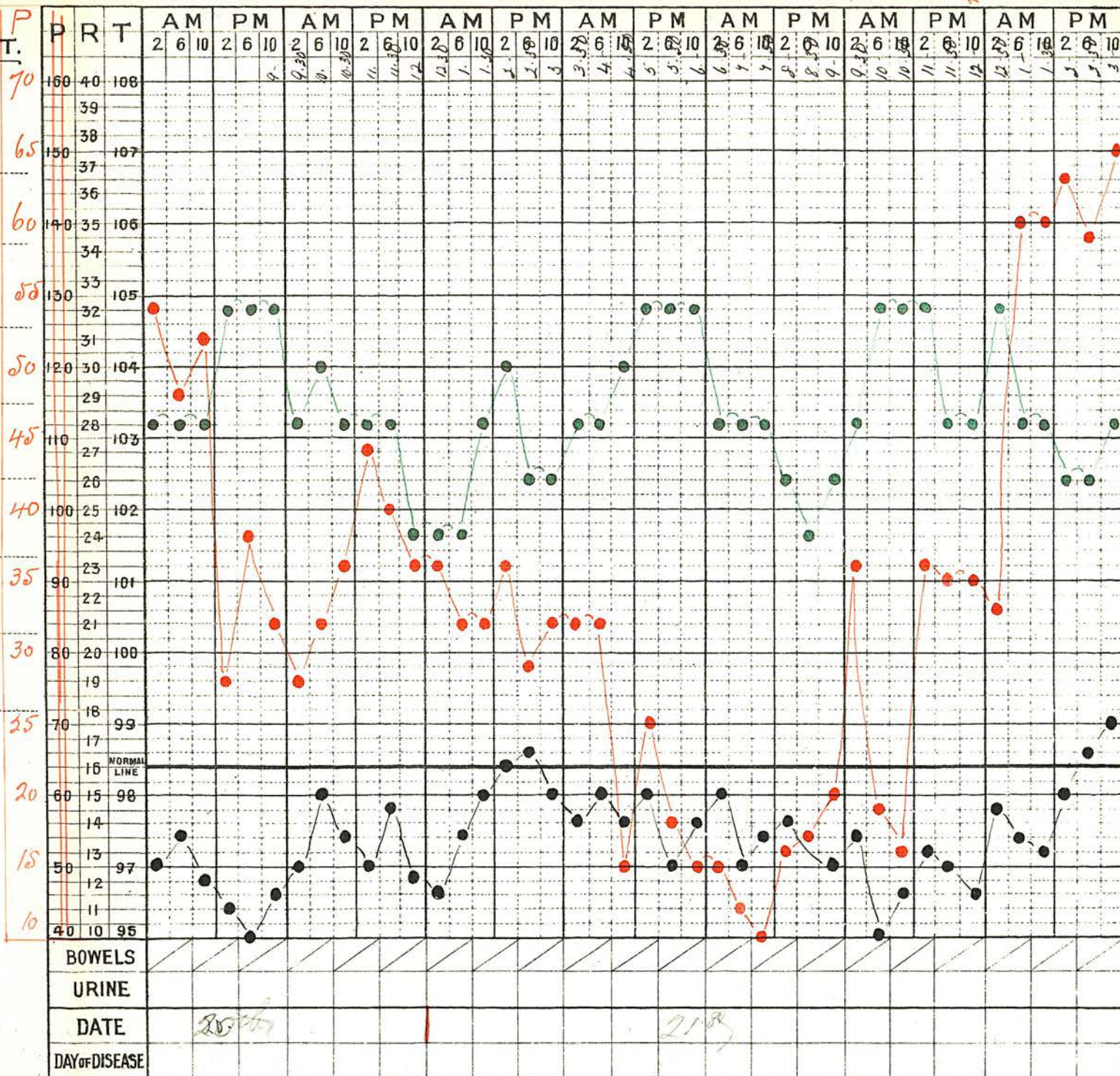
29th June, 1910

DISEASE

*Heart block
Drops*

RESULT

*Death
24th July, 1910.*



PULSE IN RED

RESPIRATION IN GREEN

TEMPERATURE IN BLACK

WALTON WORKHOUSE HOSPITAL.

4 HOURS CHART.

NAME

*Mary
Riley*

AGE

?

OCCUPATION

*House
wife*

ADMISSION

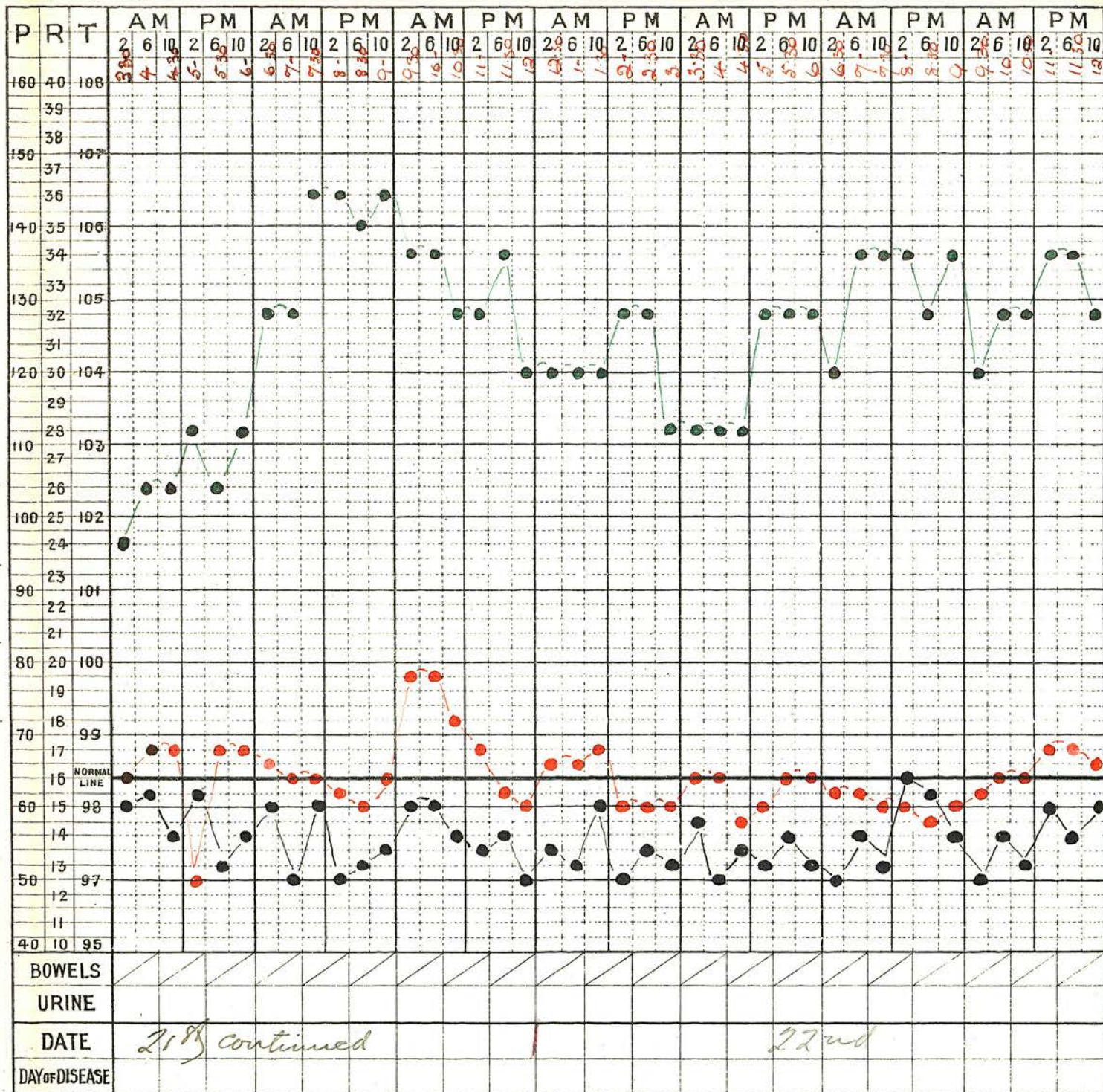
*19th June
1910*

DISEASE

Heart block

RESULT

*Death
24th July, 1910.*



PULSE IN RED

RESPIRATION IN GREEN

TEMPERATURE IN BLACK

4 HOURS CHART.

NAME _____

Mary
Kiley

AGE

7.

OCCUPATION

House-
wife

ADMISSION

29th June
1910.

DISEASE

Heart block

RESULT

SULT Death
24th July,
1910

BOWELS

URINE

DATE _____

DAY OF DISEASE

PULSE IN RED

RESPIRATION IN GREEN

TEMPERATURE IN BLACK

4 HOURS CHART.

NAME Harry
Kiley

AGE

OCCUPATION *Housewife*

ADMISSION 29th June,
1910

DISEASE Heart block

RESULT Death
24 July, 1910

BOWELS

URINE

DATE _____

DAY OF DISEASE

PULSE IN RED

RESPIRATION IN GREEN

TEMPERATURE IN BLACK

4 HOURS CHART.

NAME Mary
Riley

AGE

OCCUPATION *Housewife*

ADMISSION 29th June
1910.

DISEASE Heart block

RESULT Death
24th July, 1910.

BOWELS

URINE

DATE _____

DAY OF DISEASE

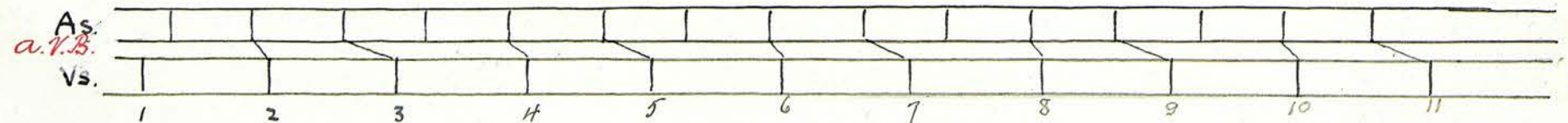
PULSE IN RED

RESPIRATION IN GREEN

TEMPERATURE IN BLACK

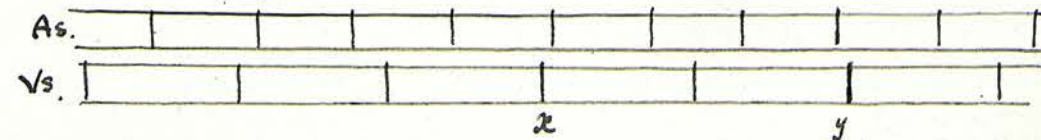
- State on admission She appears to be about 60 years of age, and is thin and anæmic in appearance.
- Circulatory system The arteries are thickened and tortuous, and the pulse rate is 33, regular in force and frequency (*vide* pulse chart). The systolic blood pressure is 150mm. Hg. Pulsations are visible in the jugular veins at the base of the neck. The apex beat is visible and palpable in the fifth left interspace. The cardiac borders are 1 inch to the right and $4\frac{1}{2}$ inches to the left of mid-sternum. There is a soft blowing systolic murmur of maximum intensity at the apex, and propagated into the axillary region. The second sound is clear and ringing and sometimes reduplicated.
- Nervous system. There is nothing to note.
- Alimentary system Normal.
- Renal system. Urine, sp. gr. 1020. The reaction is acid, and there is slight albumen present.
- Progress and treatment of the case. The patient had a light nutritious diet, and was confined to bed. She was put upon a stimulant expectorant mixture, and her condition remained unchanged until July 12th, the pulse remaining about 36 in rate and being perfectly regular in force and frequency. From July 13th to July 20th the pulse increased in frequency, varying between 43 and 48.

On July 20th a remarkable series of events happened. In the morning she complained of giddiness, and was put upon a half-hourly pulse chart. At eleven o'clock in the evening she had a slight seizure, becoming rigid but not losing consciousness; the pulse was 44. At 2 a.m. on the 21st the next seizure was noted, similar in character to the first, the pulse being now 36. The third seizure was noted at 5-30, and the pulse rate was now 18. At 7-30 a.m. the pulse dropped to 10 per minute. The writer happened to be present at the fourth and most severe seizure and took notes. At 9 a.m. he was observing the patient with his finger on the radial pulse. The first thing noted was the cessation of the radial pulse, and the patient became ashen-grey in colour; her eyes were fixed in a glassy stare upon the ceiling and her limbs were rigid; convulsive movements now attacked the whole body; the hands clenched and the shoulders jerked, whilst the breathing became stertorous and the face assumed a greyish-blue hue, and a slight froth gathered round the mouth. The radial pulse now reappeared and the patient suddenly opened her eyes, moistened her lips, and sighed once or twice. Immediately after the seizure the pulse rate was taken and it was 18 per minute. For a few minutes after the seizure she was dazed and did not understand what was

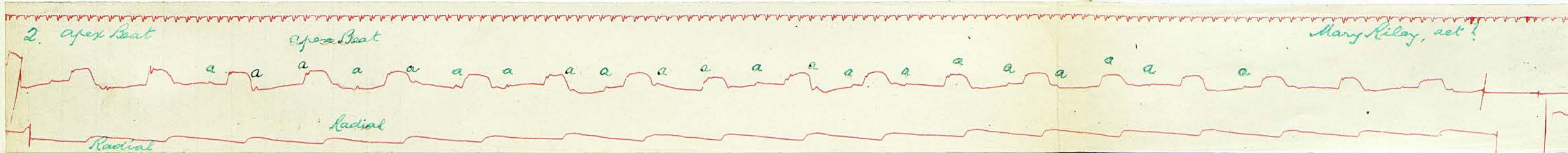
[illegible]

VS. = Ventricular Systole.

a.v.B = Auriculo-Ventricular Bundle



Partial, (complete?), Heart-block.

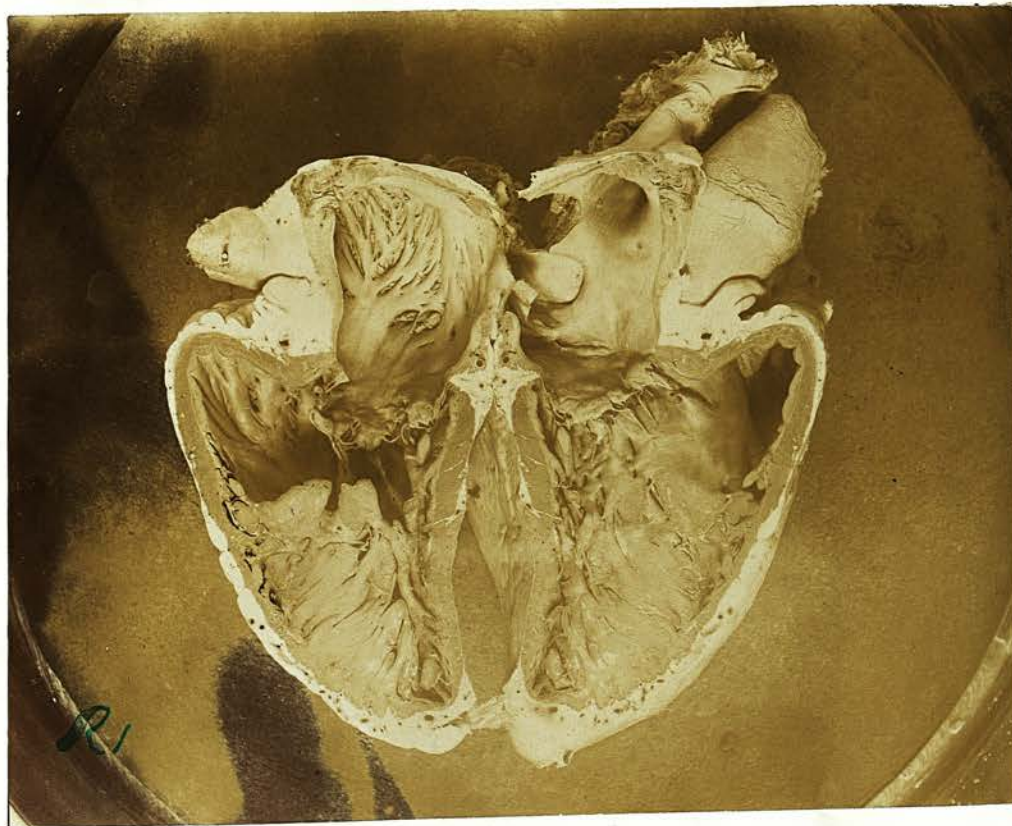


R2. Left heart



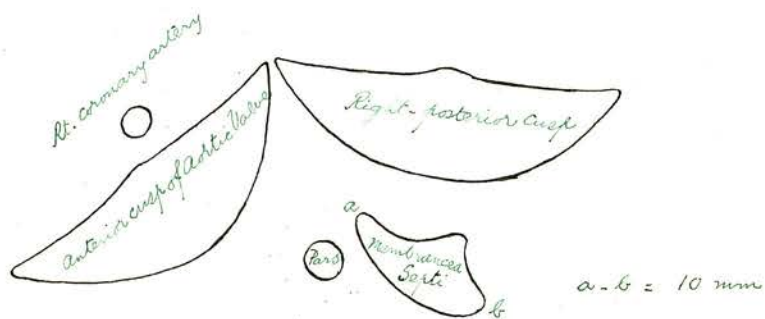
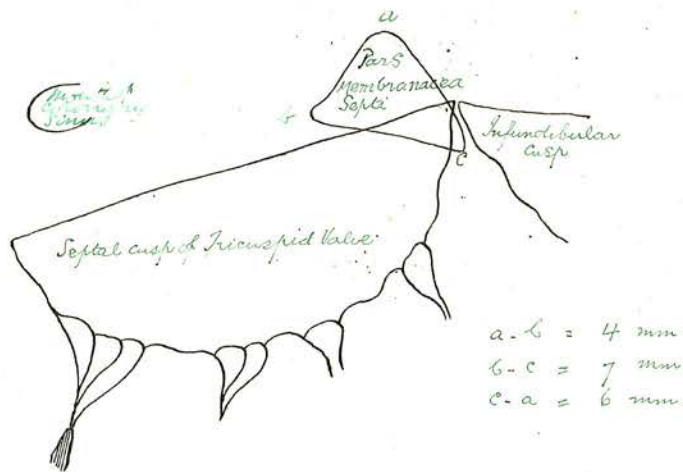
Mary Riley.

MARY RILEY



R 1. Right Heart.

Mary Riley



Diagrams to shew the relation of the
pars membranacea septi to the
tricuspid and aortic valves.

Seen from the right side the pars membranacea septi is a small, relatively opaque, triangular area, the sides of which measure 6.4 and 7mm., respectively (see diagram.)

- Tricuspid orifice. The tricuspid orifice is 9.7cm. in circumference. The free margins of the valve segments are thickened. The chordæ tendineæ are not thick or shrunk, and the papillary muscles are not fibrous.
- Right ventricle. The columnæ carneæ are markedly hypertrophied, and the ventricular muscle presents a few small fibrous areas.
- Pulmonary orifice. The pulmonary orifice is 7cm. in circumference. The pulmonary valve and artery are healthy.
- Left auricle. The endocardium is markedly thick, and the muscle wall is hypertrophied.
- Mitral orifice. The mitral orifice is 8.2cm. in circumference. The marginal cusp is short but not thick. The aortic cusp is not shrunk, being 21mm. from apex to mid-point of the line of attachment. The chordæ tendineæ are not thick. In the papillary muscles there are small fibrous patches.
- Left ventricle. The musculature is hypertrophied. A considerable degree of fibrous change is seen in the muscle wall.
- Aortic orifice. The aortic orifice is 6cm. in circumference. The anterior and right-posterior cusps are slightly thickened, while the left posterior cusp is calcareous. In the aortic wall above the sinuses of valsalva there are nodular plaques of atheroma, and 6cm. above the free margins of the aortic cusps there is some syphilitic cicatrization of the aortic wall. The walls of the coronary arteries present calcareous patches of atheroma.

CASE 5.

Heart-block without History of Adams-Stokes Syndrome.

FRANK CAMPBELL, Aet. 67

- Date of admission. July 19th, 1908.
- Complaint. Chronic bronchitis.
- General history. On admission pulse was 66, respirations 24, and temperature 97.8. He stated that he had been a brick-field worker for 30 years and that his work had always been hard. He suffered from acute bronchitis 30 years ago, has had gonorrhœa a good many times, and also admits having syphilis; there are syphilitic scars on the body. Patient remained an in-patient, being treated for bronchitis and emphysema, and his history was uneventful until the 5th September, 1910.

WALTON WORKHOUSE HOSPITAL.

4 HOURS CHART.

NAME

Frank Campbell

AGE

67 years

OCCUPATION

Dock Labourer

ADMISSION

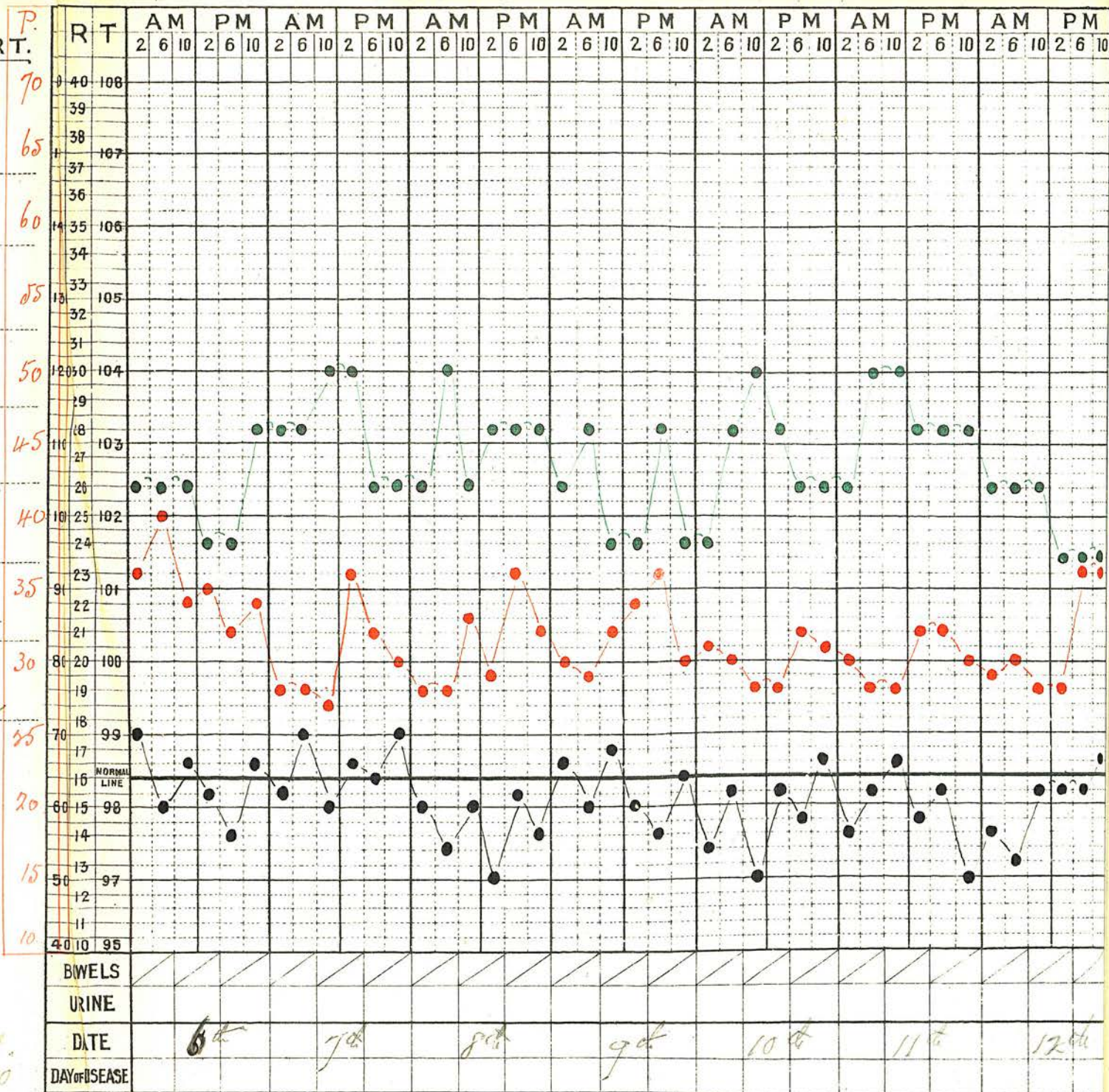
18th July 1906

DISEASE

Heart block

RESULT

Death 20th November 1910



PULSE IN RED

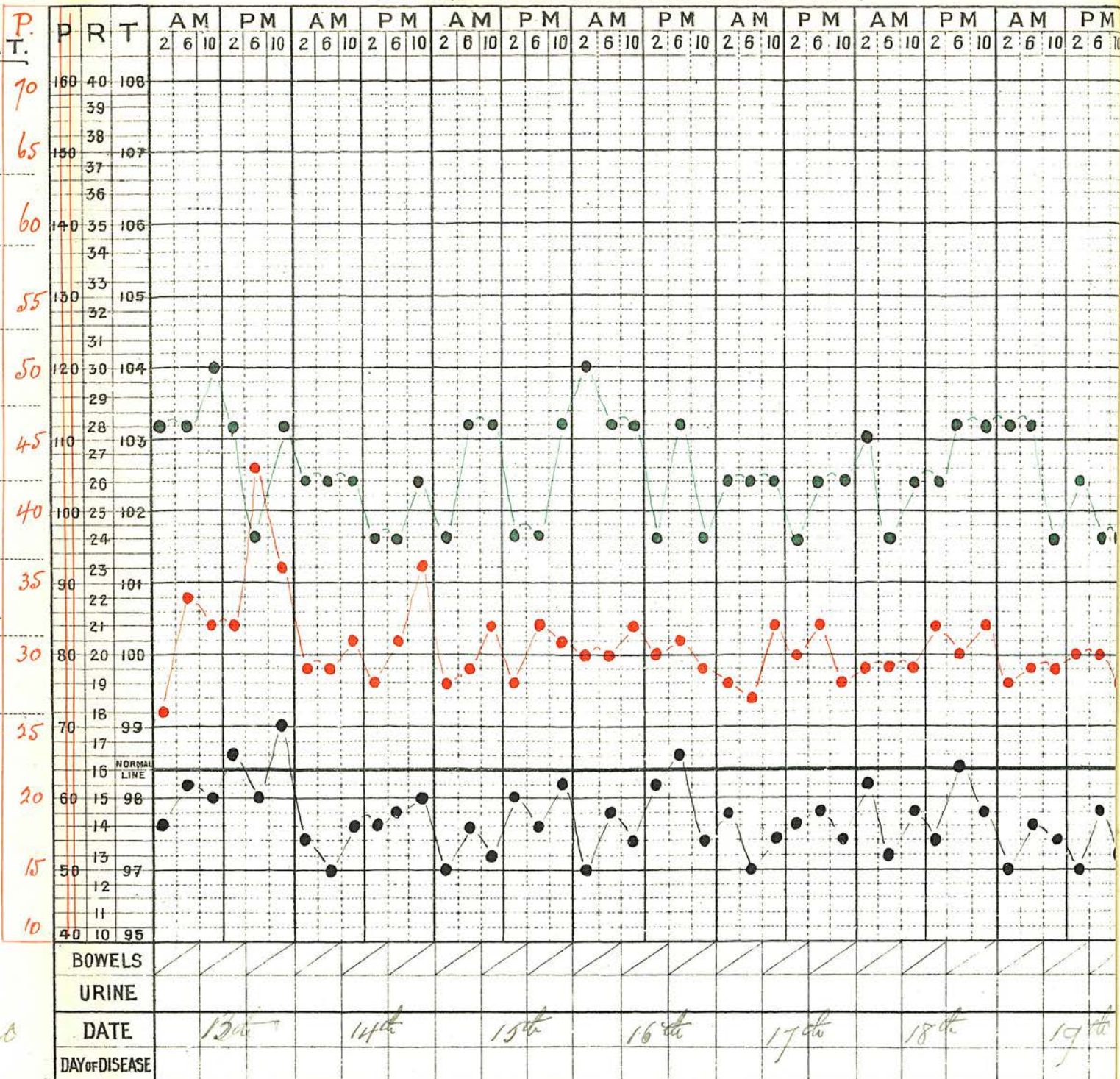
RESPIRATION IN GREEN

TEMPERATURE IN BLACK

No 2

WALTON WORKHOUSE HOSPITAL.

4 HOURS CHART.

NAME *Frank
Campbell*AGE *67 years*OCCUPATION *Dock
Labourer*ADMISSION *18th July
1908*DISEASE *Heart block*RESULT *Death
20th November, 1910*

PULSE IN RED

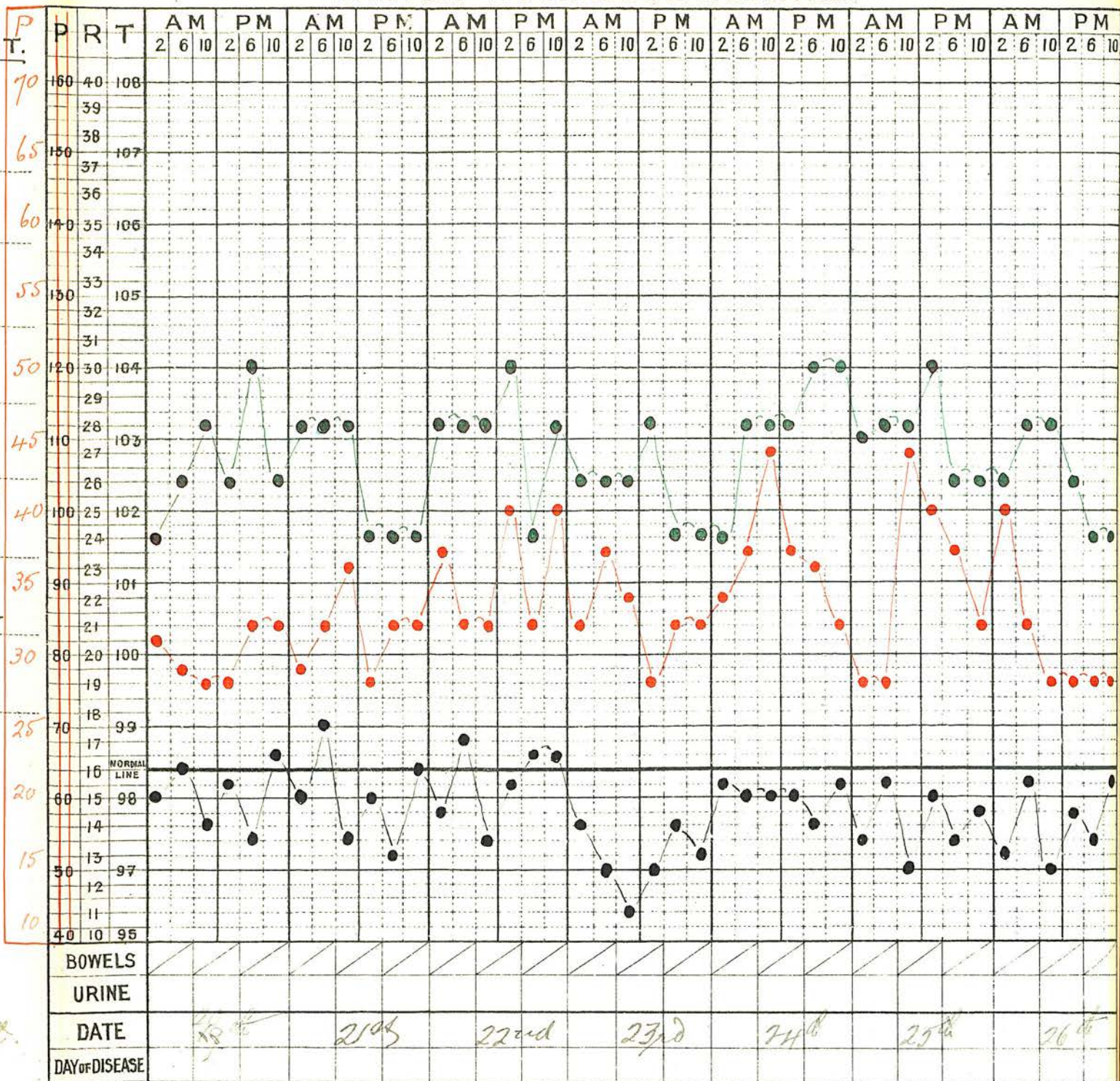
RESPIRATION IN GREEN

TEMPERATURE IN BLACK

No. 3

WALTON WORKHOUSE HOSPITAL.

4 HOURS CHART.

NAME *Frank Campbell*AGE *67 years*OCCUPATION *Dock Labourer*ADMISSION *18th July, 1908*DISEASE *Heart block*RESULT *Death*
20th November, 1910.

WALTON WORKHOUSE HOSPITAL.

4 HOURS CHART.

NAME

Frank Campbell

AGE

67 years

OCCUPATION

Dock Labourer

ADMISSION

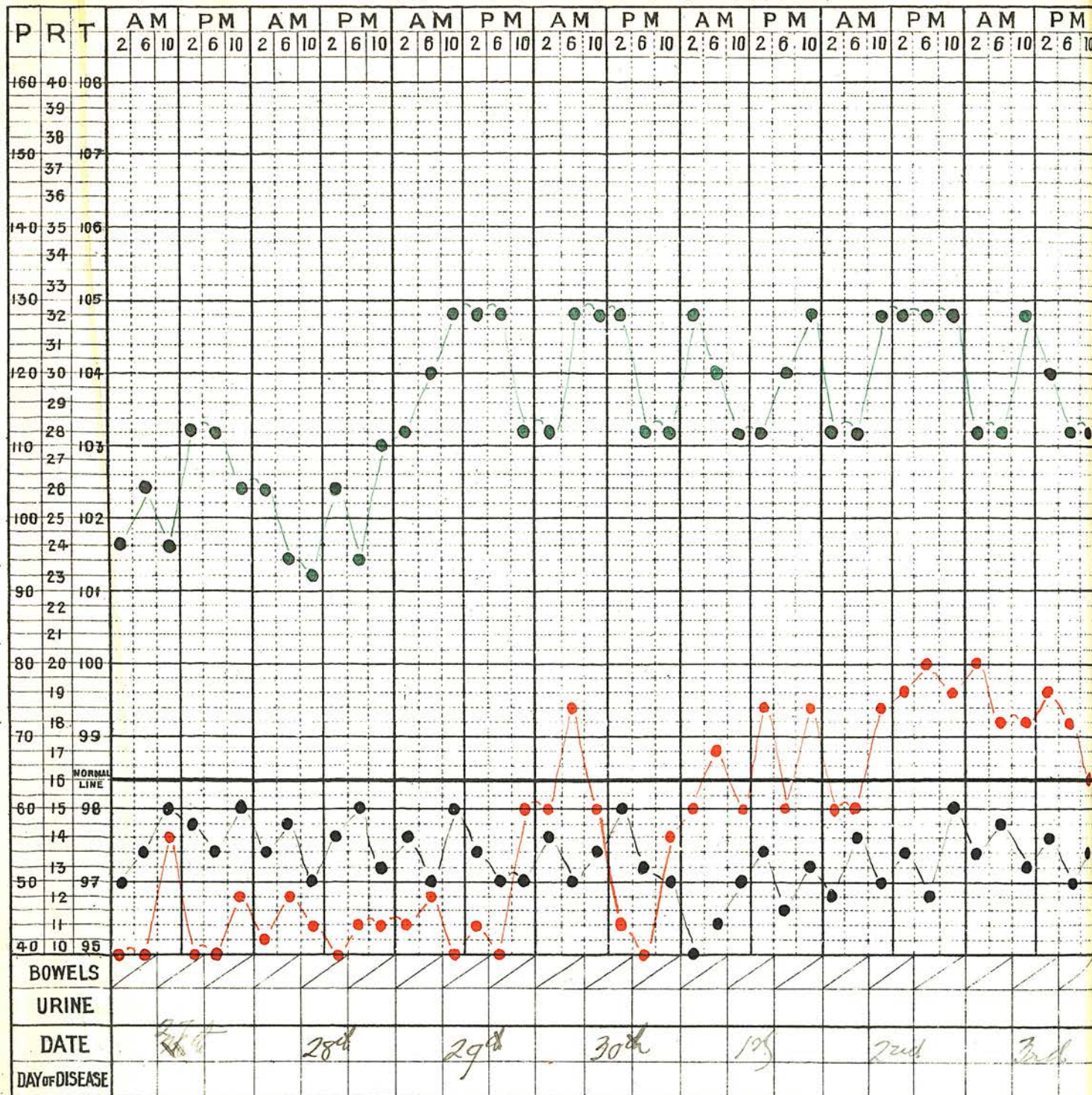
18th July, 1908

DISEASE

Heart block

RESULT

*Death
20th November, 1910*



PULSE IN RED

RESPIRATION IN GREEN

TEMPERATURE IN BLACK

NAME _____

Frank
Campbell

AGE

67 years

OCCUPATION

Doek
Labourer

ADMISSION

18th July
1908

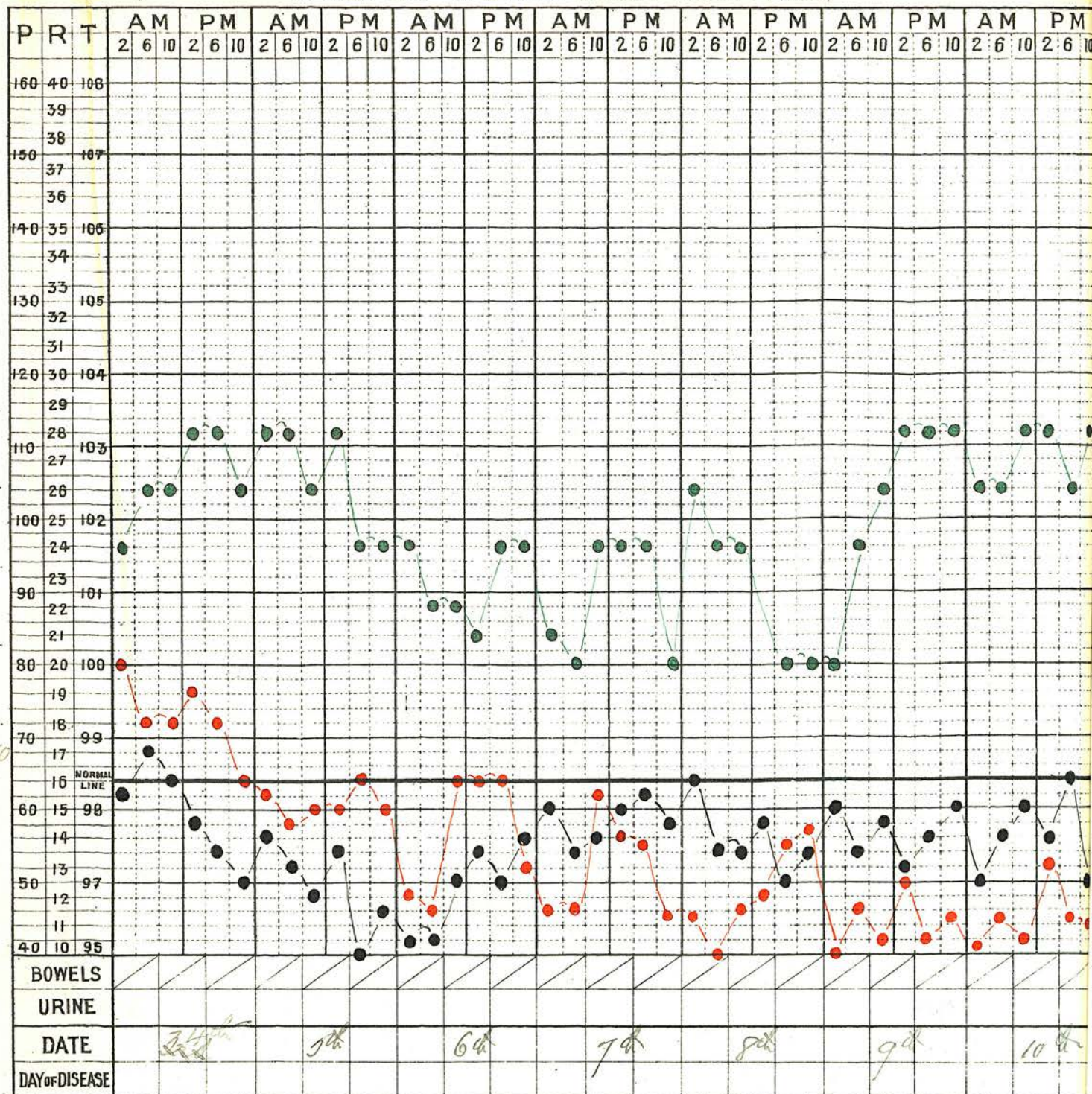
DISEASE

Heart block

RESULT

Death

20th November, 1910



PULSE IN RED

RESPIRATION IN GREEN

TEMPERATURE IN BLACK

No 6

WALTON WORKHOUSE HOSPITAL.

4 HOURS CHART.

NAME

Frank
Campbell

AGE

67 years

OCCUPATION

Dock
Labourer

ADMISSION

18th July,
1908

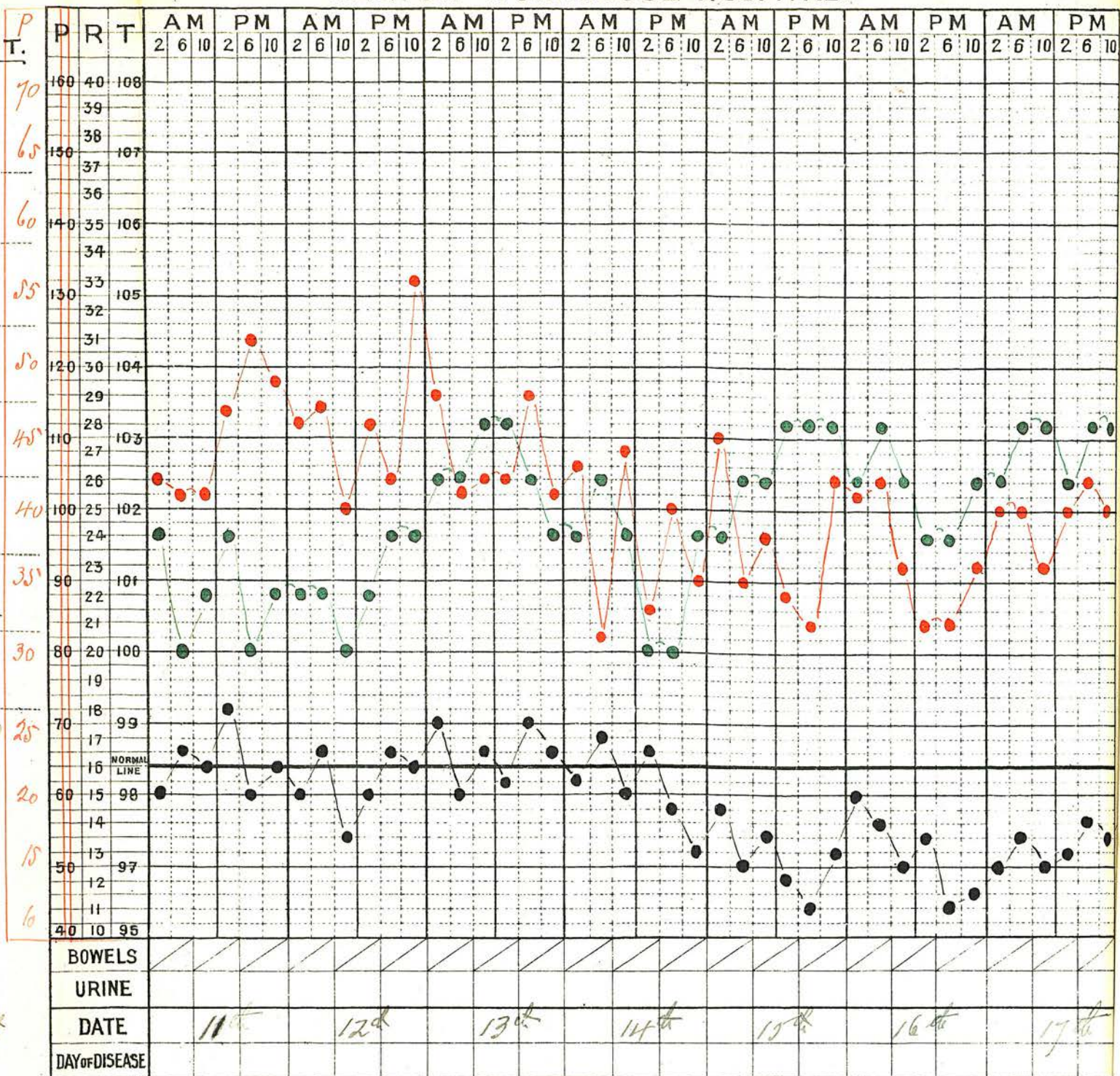
DISEASE

Heartblock

RESULT

Death

20th November, 1910



PULSE IN RED

RESPIRATION IN GREEN

TEMPERATURE IN BLACK

WALTON WORKHOUSE HOSPITAL.

4 HOURS CHART.

NAME *Frank Campbell*

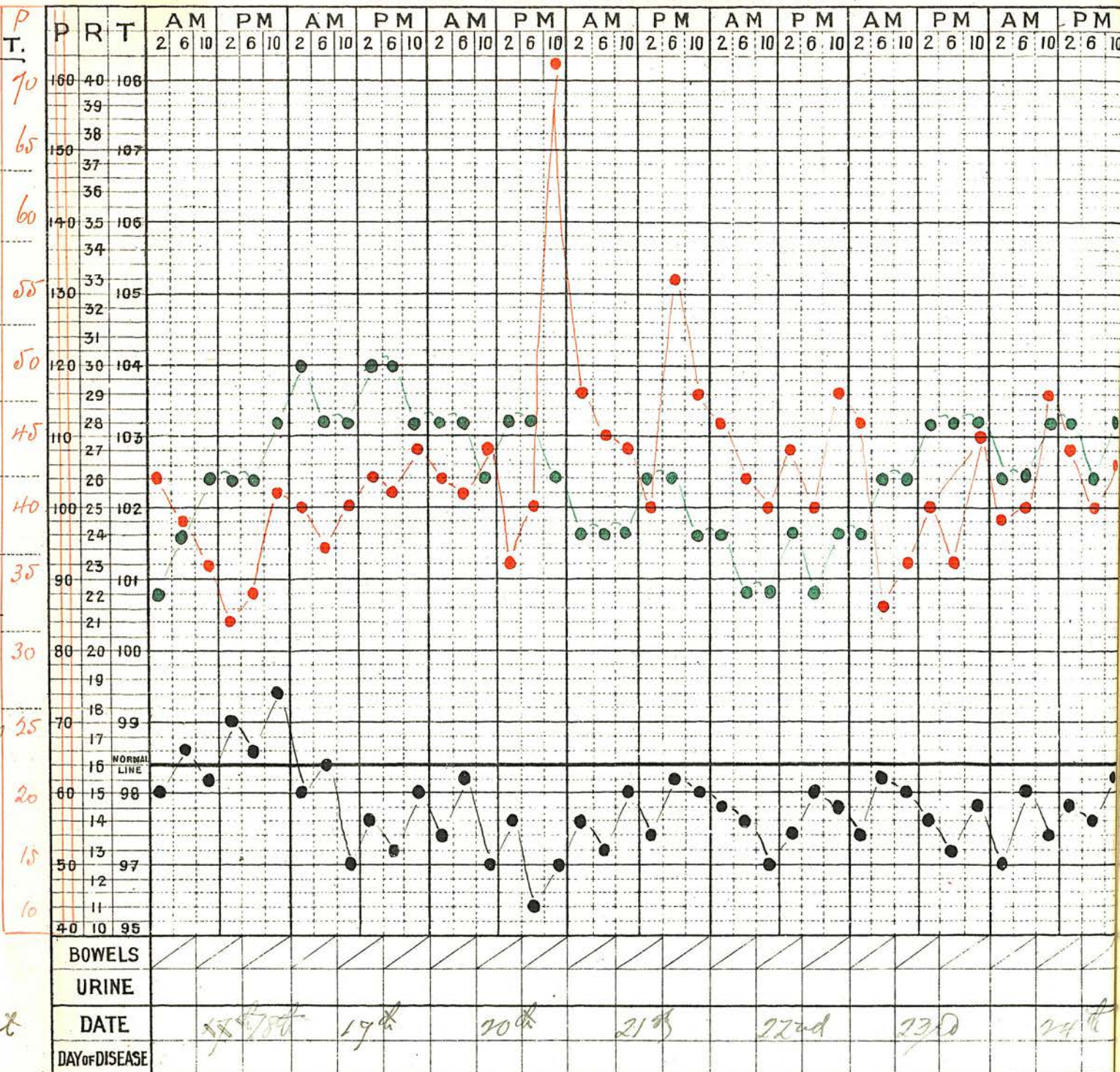
AGE *67 years*

OCCUPATION *Dock Labourer*

ADMISSION *18th July, 1908*

DISEASE *Heart Block*

RESULT *Death*
20th November, 1910



PULSE IN RED

RESPIRATION IN GREEN

TEMPERATURE IN BLACK

WALTON WORKHOUSE HOSPITAL.

4 HOURS CHART.

NAME

Frank
Campbell

AGE

67 years

OCCUPATION

Dock Labourer
18th July 1908

ADMISSION

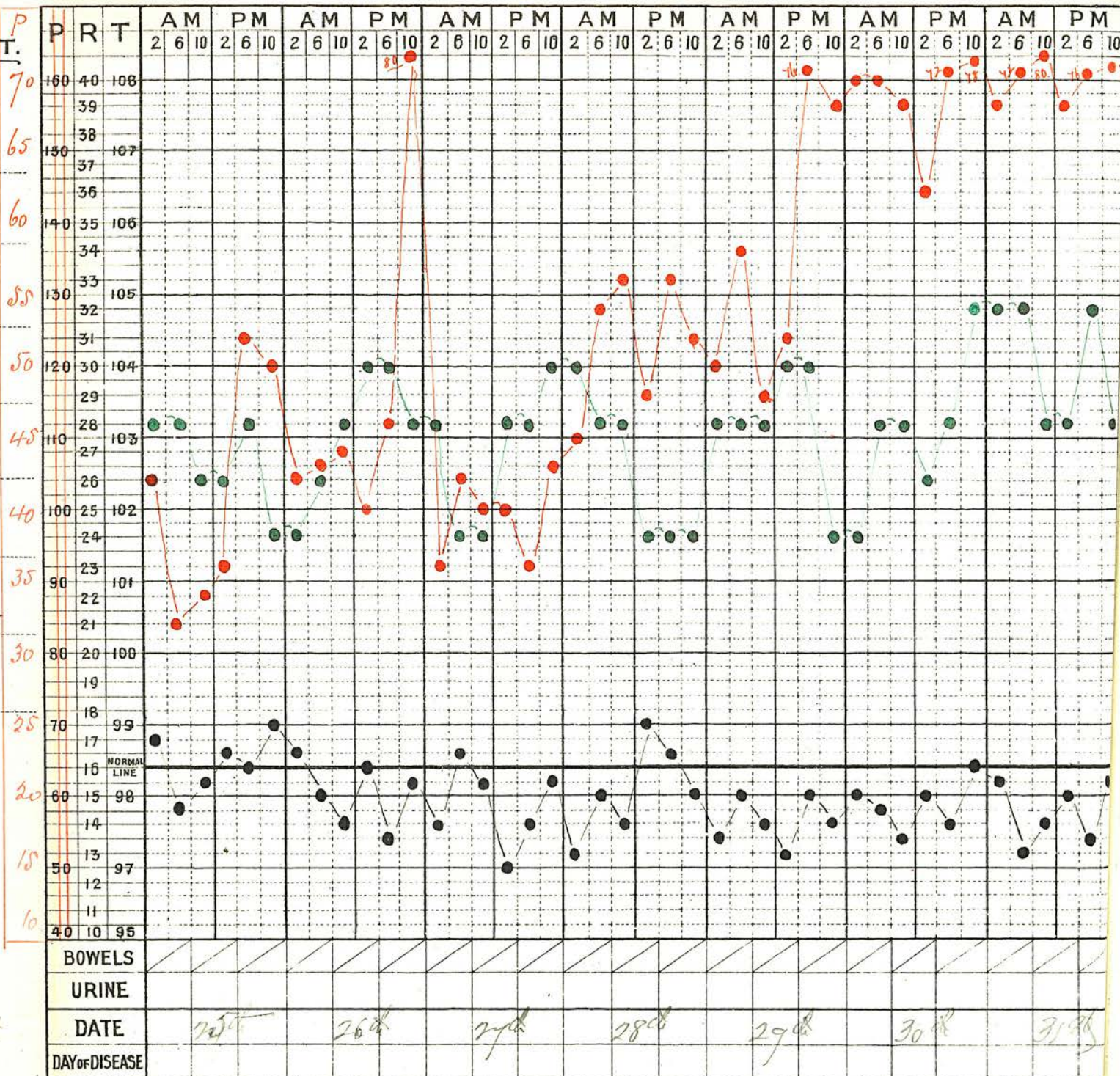
18th July 1908

DISEASE

Heart block

RESULT

Death
20th November,
1910



PULSE IN RED

RESPIRATION IN GREEN

TEMPERATURE IN BLACK

WALTON WORKHOUSE HOSPITAL.

4 HOURS CHART.

NAME

Frank Campbell

AGE

67 years

OCCUPATION

Dock Labourer

ADMISSION

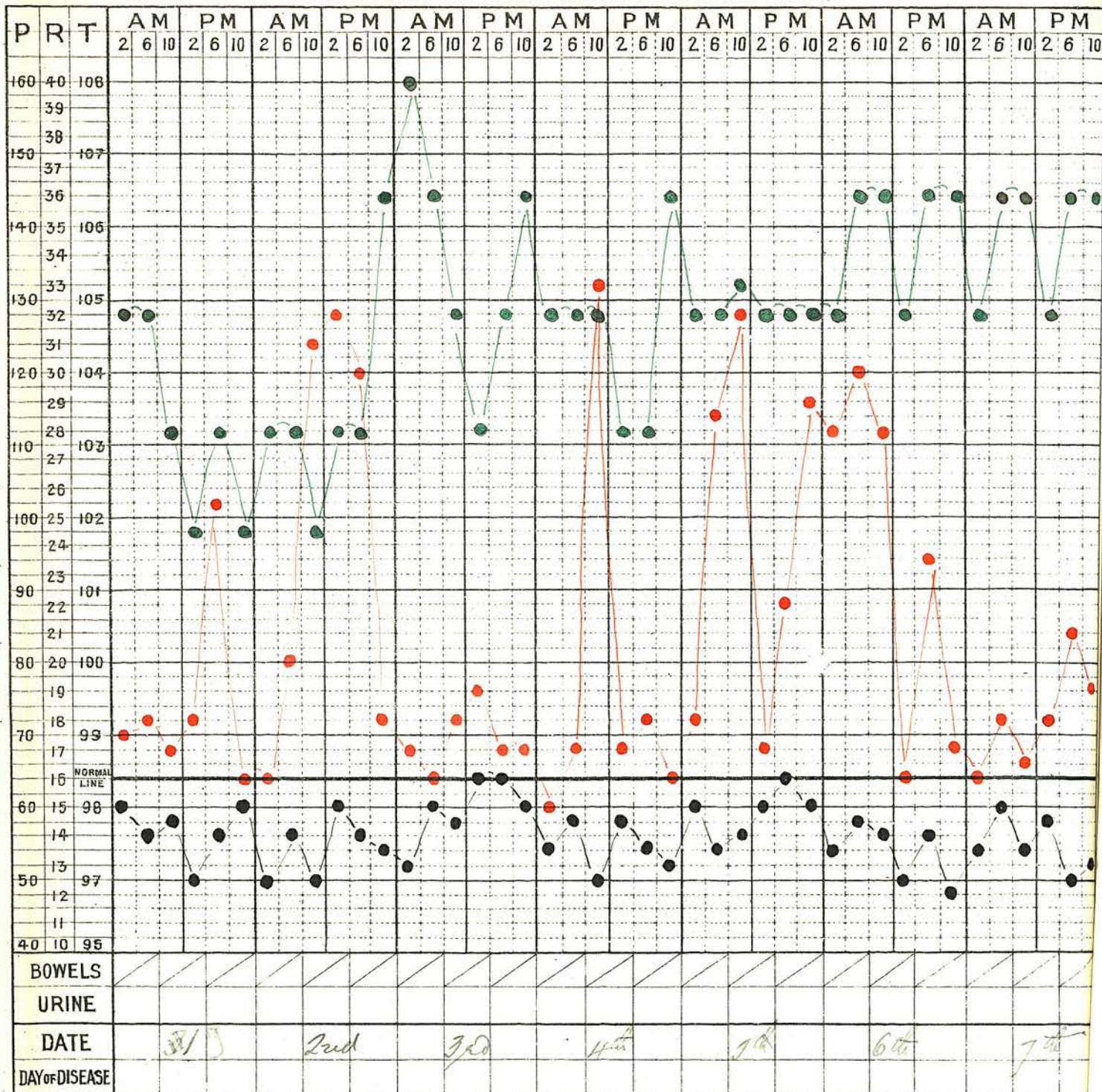
18th July, 1908

DISEASE

Heart block

RESULT

*Death
20th November,
1910*



PULSE IN RED

RESPIRATION IN GREEN

TEMPERATURE IN BLACK

No. 10

WALTON WORKHOUSE HOSPITAL.

4 HOURS CHART.

NAME

Frank
Campbell

AGE

67 years

OCCUPATION

Dock
Labourer

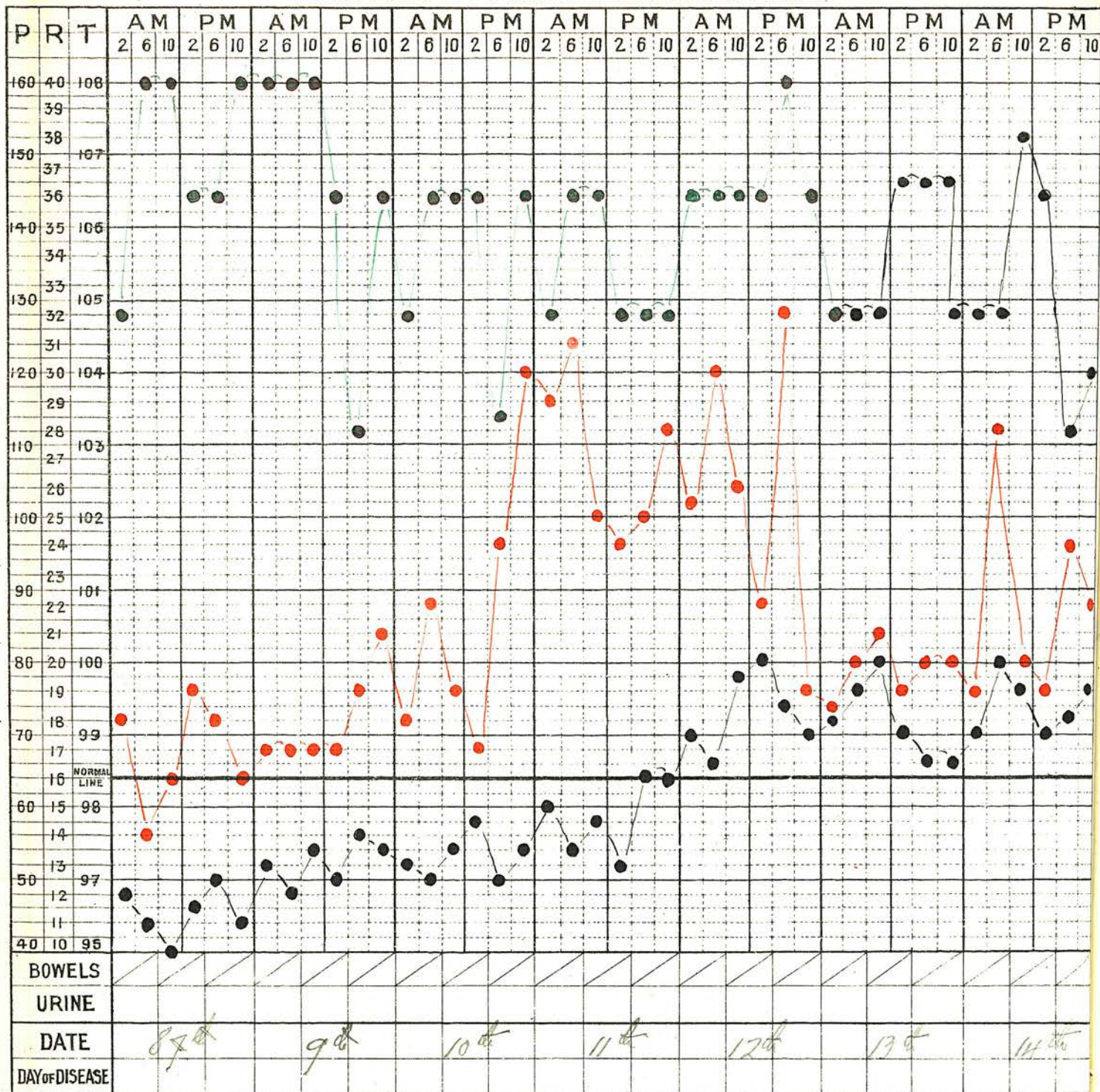
ADMISSION

18th July,
1908

DISEASE

Heart block

RESULT

Death
20th November,
1910

PULSE IN RED

RESPIRATION IN GREEN

TEMPERATURE IN BLACK

No 11

WALTON WORKHOUSE HOSPITAL.

4 HOURS CHART.

NAME *Frank Campbell*

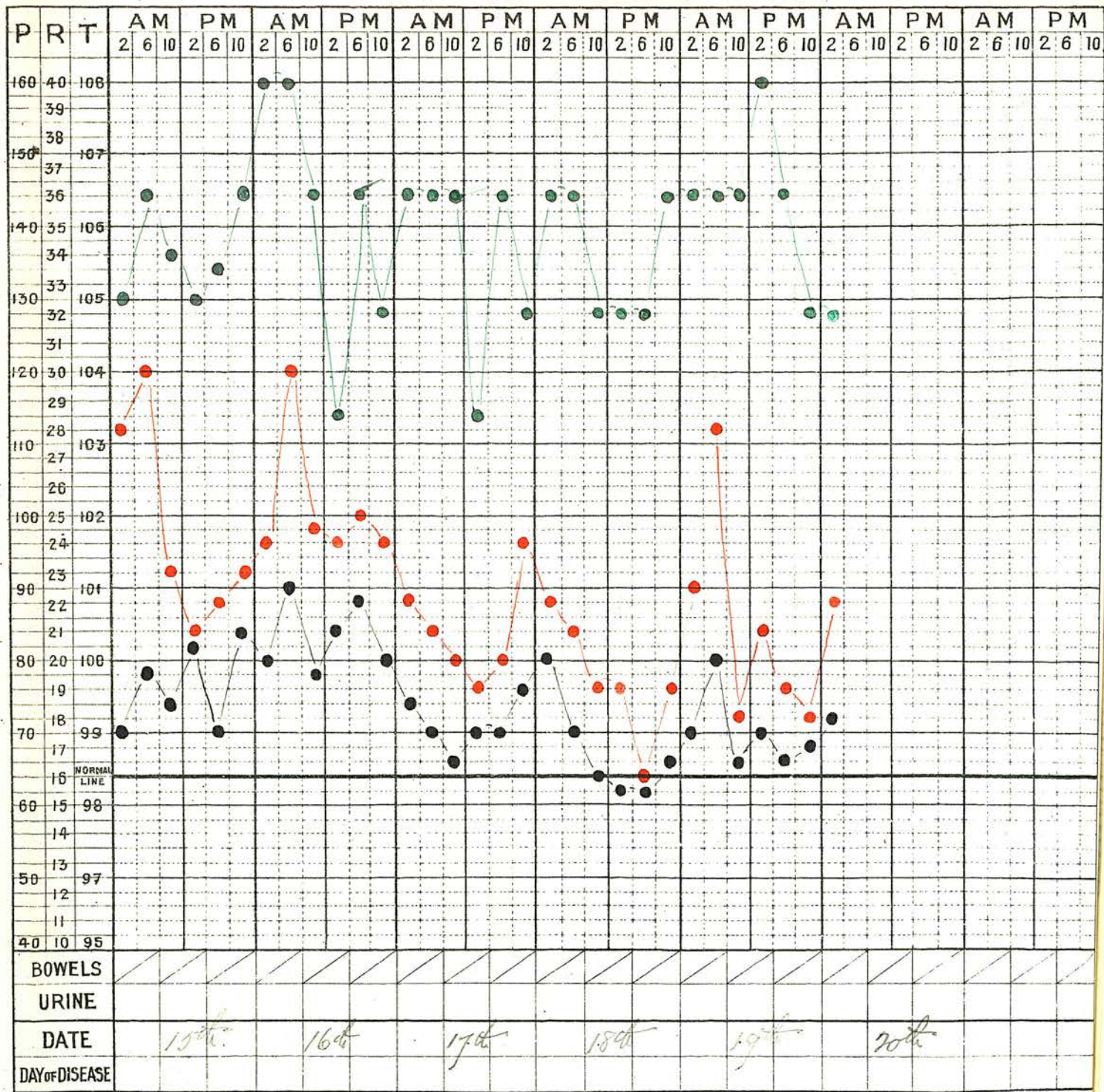
AGE *67 years*

OCCUPATION *Dock Labourer*

ADMISSION *18th July, 1908*

DISEASE

RESULT *Death*
20th November, 1910



PULSE IN RED RESPIRATION IN GREEN TEMPERATURE IN BLACK

C3.

Longitudinal sections
of left auricle and
ventricle to show the
relation of the calcareous
ring in the basal portion
of the ventricular muscle
to the marginal cusp of
the mitral valve.



C3.

Fig 1. The line of
section passes
through the mid-
point of line of
attachment of the
marginal cusp.

Fig 2. The line of
section passes mid-
way between mid-
point and inner-
end of line of
attachment of the
cusp.

Fig 3. Near inner
end of line of
attachment of the
cusp.

C4. Mass excised from central
portion of heart, viewed from
front and left side



1. Portion of anterior cusp of Aortic Valve
2. Deepest part of Right-posterior
cusp of Aortic valve
3. Pars membranacea septi
4. Aortic cusp of Mitral valve.

Between 3 and 4 there are a
number of calcareous nodules
projecting beneath the endo-
cardium.

C5. Mass excised from central
portion of the heart, viewed
from behind

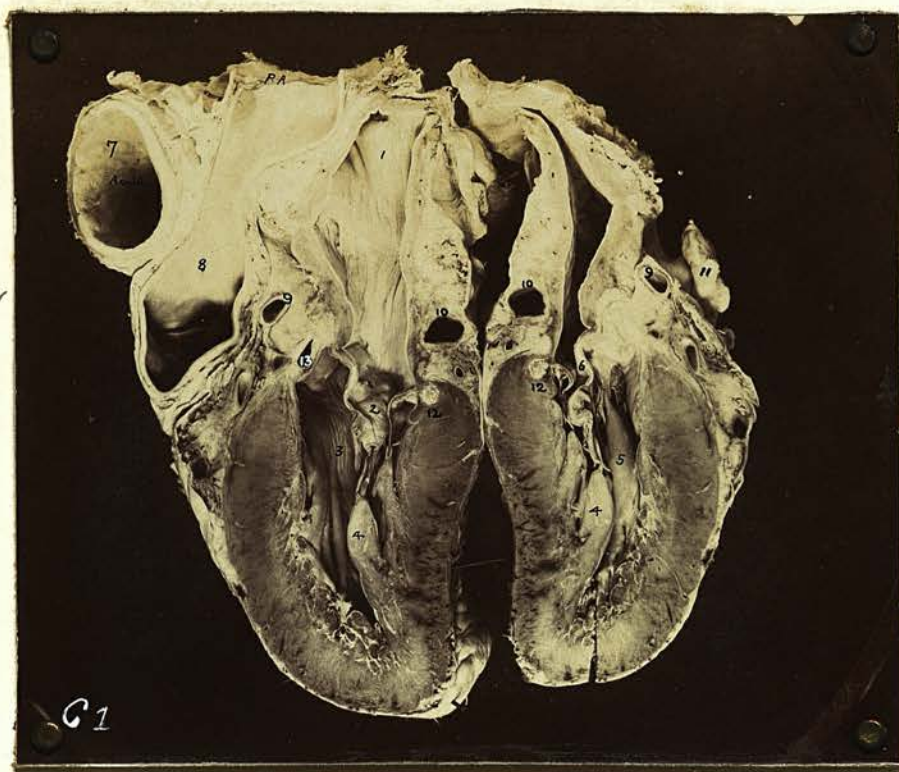


1. Portion of Anterior cusp of Aortic valve
2. Right posterior Sinus of valsalva.
3. Septal wall of left ventricle below
pars membranacea septi
4. Thick Aortic cusp of Mitral valve with
calcareous nodules projecting on its
auricular surface
5. Auricular septal wall
6. The numeral is placed just above the
transverse section of the calcareous ring.

Frank Campbell

C1.

1. Septal wall of left auricle
2. Aortic cusp of mitral valve
3. Septal wall of left ventricle
4. Posterior papillary muscle
5. Anterior papillary muscle
6. Marginal cusp of mitral valve
7. Aorta

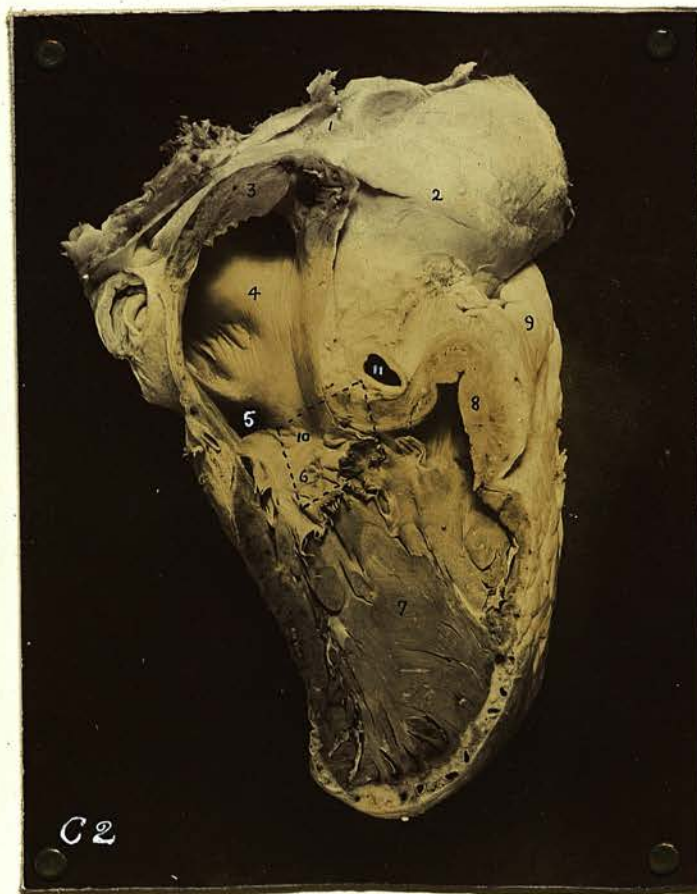


C1.

8. Pulmonary artery
9. Left coronary artery
10. Coronary sinus
11. Left atricular appendix
12. Calcaneous ring
13. Left posterior sinus of Valsalva.

C2.

1. Superior vena cava
2. Aorta
3. Atrioventricular septum
4. Septal wall of right auricle
5. Mouth of coronary sinus
6. Septal cusp of tricuspid valve



C2.

7. Ventricular septum
8. Wall of conus arteriosus
9. Pulmonary artery
10. Puckered depression on septal wall of auricle
11. Anterior sinus of Valsalva.

Frank Campbell.

Onset of Brady- cardia.	On September 6th his pulse dropped to 34, and the following day it had dropped to 27, otherwise there being no change in his condition. He stated that he had had no fits or fainting attacks, and that he felt just as usual.
Circulatory system	There is general oedema of the body. The radial arteries are hard and tortuous; the pulse varies in rate between 25 and 35, and it is regular in force and frequency (<i>vide</i> pulse chart). The systolic blood pressure is 200mm. of Hg. Pulsations are visible in the jugular veins. The apex beat is in the fifth interspace, just outside the nipple line. The right border is $2\frac{1}{2}$ inches to the right, and the left is 5 inches to the left of mid-sternum. There is a soft systolic murmur at the apex, and the second sound is closed.
Respiratory system	The chest is barrel-shaped, and the lungs are emphysematous, encroaching on the heart and bulging into the neck. There are high-pitched and sonorous rhonchi all over the chest.
Nervous system.	There is nothing to note.
Digestive system.	The liver is enlarged.
Renal system.	Urine, sp. gr. 1020. The reaction is acid, and there is no albumen.
Progress and treatment of the case.	The records of the pulse began on September 5th, 1910; the rate was then between 28 and 35.

The bronchitis and emphysema were troubling him, and his treatment consisted of strychnine gr. $\frac{1}{60}$ four-hourly, and brandy half-an-ounce three-hourly. A cough mixture was also given. He had very bad attacks of dyspnoea of an asthmatic type. On September 22nd the pulse became quicker, being 40; on September 24th it was 44, and on September 27th it increased to 55. From the 30th September until the 4th October the rate was between 60 and 80. The patient's breathing was so embarrassed that it was impossible to record accurately the jugular pulsations. From October 6th until October 27th, with two exceptions, the pulse rate was between 32 and 55; on the 20th it was 73, and on the 26th 80. From the 29th October until death it varied between 60 and 132 (*vide* pulse chart). He gradually became weaker and died on the 20th of November.

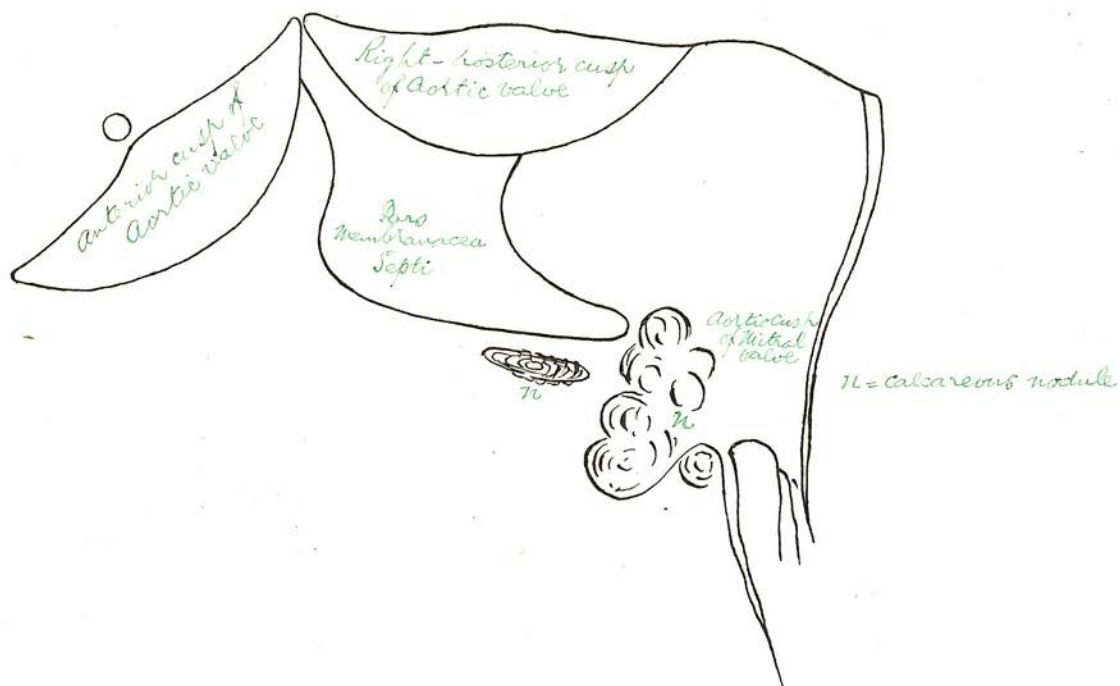
The patient had no seizures or syncopal attacks.

No graphic records of the case were taken.

Pathological Examination of the Heart.

Heart.	The heart weighs 525 grammes.
External measurements.	Circumferential measurement in the long axis, 36cm.; circumferential measurement around the auriculo-ventricular groove, 30.5cm.; length, 15cm.; breadth, 11.5 cm.; antero-posteriorly, 7.2cm.

Frank Campbell



Calcareous masses in relation to
the paraventricular septum

- Long diameters of the cavities. Right auricle, 6.5cm.; right ventricle, 7.0cm.; left auricle, 7.5cm.; left ventricle, 7.3cm.
- Epicardium. The epicardium is thickened in the form of a large "milk-spot" over the whole of the right auricle, and there is a large "milk-spot" over the middle of the right ventricle. There is a considerable deposit of adipose tissue over the right auricle and over the conus arteriosus.
- Right auricle. Over the whole cavity the endocardium is somewhat thick. The muscle wall is hypertrophied, especially the *tænia terminalis* and the pectinate muscles. No pathological change can be detected at the junction of the superior vena cava and auricle. The foramen ovale is closed. Viewed from the right side the *pars membranacea septi* is of triangular form, semi-opaque and thick. On the septal wall behind the lower posterior angle of the *pars membranacea septi* there is a puckered depression. The endocardium at this point is not specially thickened. The auricular septum behind the *pars membranacea septi* is felt to be thick and hard.
- Tricuspid orifice. It is 10.5cm. in circumference. The valve segments are all healthy, and the chordæ tendineæ are not thick or shrunken.
- Right ventricle. The musculature is not hypertrophied.
- Pulmonary orifice. The pulmonary orifice is 7.5cm. in circumference.
The pulmonary valve and artery are healthy.
- Left auricle. The endocardium is somewhat thickened. The musculature is hypertrophied.
- Mitral orifice. The mitral orifice at the line of attachment of the valve is 9.9cm. in circumference. The marginal cusp is thick, but not shrunken. The basal part of the cusp and the adjacent muscle are involved by the calcareous ring which will be described later. The aortic cusp of the mitral valve is markedly thick, and in its central and basal portions there are nodular calcareous bosses projecting upon both the auricular and ventricular surfaces. The chordæ tendineæ are but little thickened, and they are not shrunken, but the apical portions of the papillary muscles are fibrous.
- Left ventricle. The muscle wall is 1.8cm. thick at the level of the apices of the papillary muscles, and 1.2cm. thick at the apex of the chamber. The muscle does not present to the naked eye any evidence of fibrosis.
- Aortic orifice. The aortic orifice is 7cm. in circumference. The basal portion of all the cusps, and the adjacent portions of the sinuses of *valsalva*, are thick and fibrous, but they are not calcareous. There is, however, a small calcareous nodule projecting upon the endocardium below the deepest part of the attached margin of the anterior cusp (see X-ray photograph and diagram).

Frank Campbell
Posterior

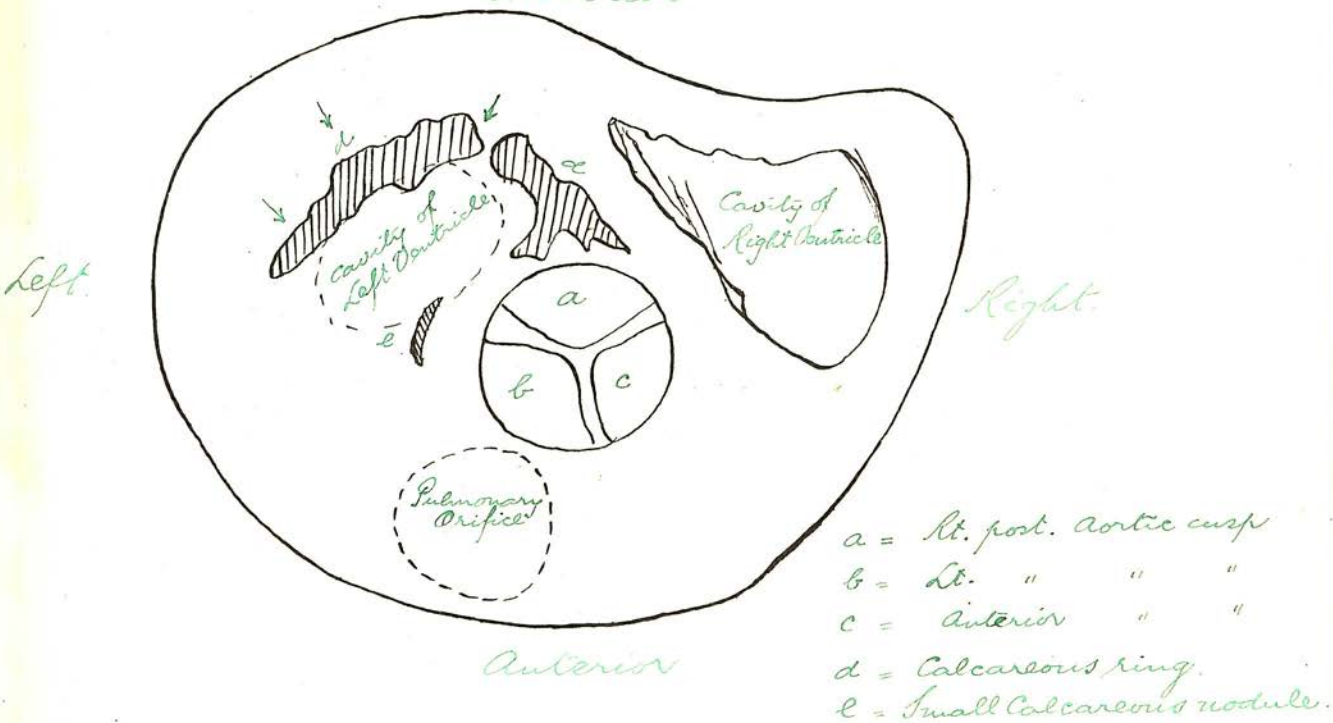


Anterior
X-Ray photograph showing the Calcareous ring.

Frank Campbell

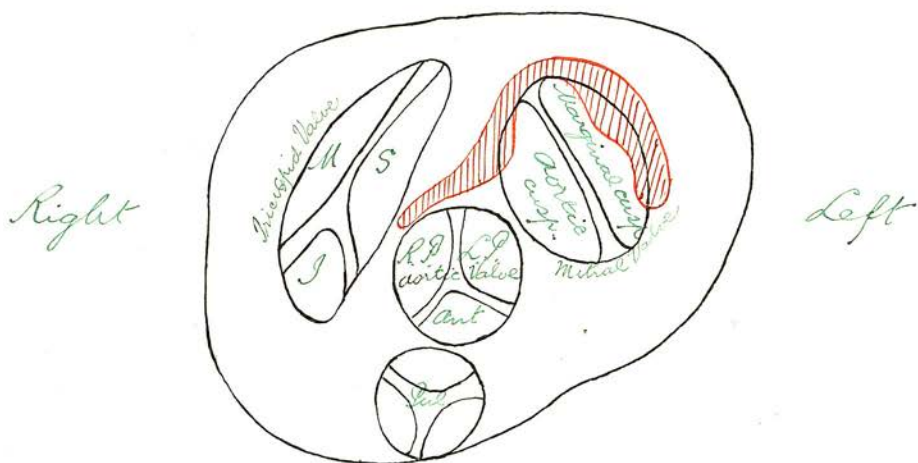
Diagram of the
X-Ray photograph

Posterior



The heart had been considerably cut up,
and was pieced together later for the
purpose of taking the X-ray photograph,
so the ring is cut transversely in these
places - ↓.

Frank Campbell



The Calcareous ring in red

The wall of the ascending aorta presents a few small patches of atheroma, and similar patches are seen in the walls of the coronary arteries and of their branches.

The calcareous ring.

Lying in the basal part of the musculature of the left ventricle, and in the plane of the mitral orifice, there is a calcareous, incomplete ring. It begins, (see X-ray photograph), just internal to the outer end of the marginal cusp of the mitral valve, and passes round in the ventricular muscle close to the line of attachment of this cusp. The calcareous ring then projects into the marginal cusp, forming nodular masses upon both the auricular and ventricular surfaces of this cusp. On transverse section at the mid-point of the line of attachment of the marginal cusp, (fig. 3 photograph), the calcareous ring is ovoid and 8mm. by 4mm., the longer diameter being in the long axis of the heart and at a right angle to the plane in which the ring lies. Midway between the mid-point and the inner end of the attached margin of the marginal cusp, (fig. 2, photograph), the ring is even thicker, for the ovoid transverse section measures 8.5mm. by 5mm. Further towards the inner end of the line of attachment of the marginal cusp, the ring becomes thinner, and here, (see photograph fig. 1), the ring is surrounded on all sides by ventricular muscle, although the layer of muscle separating the ring from the attached margin of the cusp is 1mm. thick.

The ring then passes around in the basal part of the inner third of the aortic cusp of the mitral valve, and thereafter passes obliquely forwards and to the right in the adjacent muscle of the ventricular septum. This portion of the ring forms a number of irregular, nodular bosses upon both the auricular and ventricular surfaces of the basal part of the aortic cusp of the mitral valve (photograph). The largest of these bosses projects partly from the ventricular surface at the line of attachment of the cusp, and partly from the adjacent portion of the ventricular septum, behind and below the posterior angle of the pars membranacea septi (photograph), where the ring terminates at a point 7mm. below the mid-point of the line of attachment of the right-posterior cusp of the aortic valve.

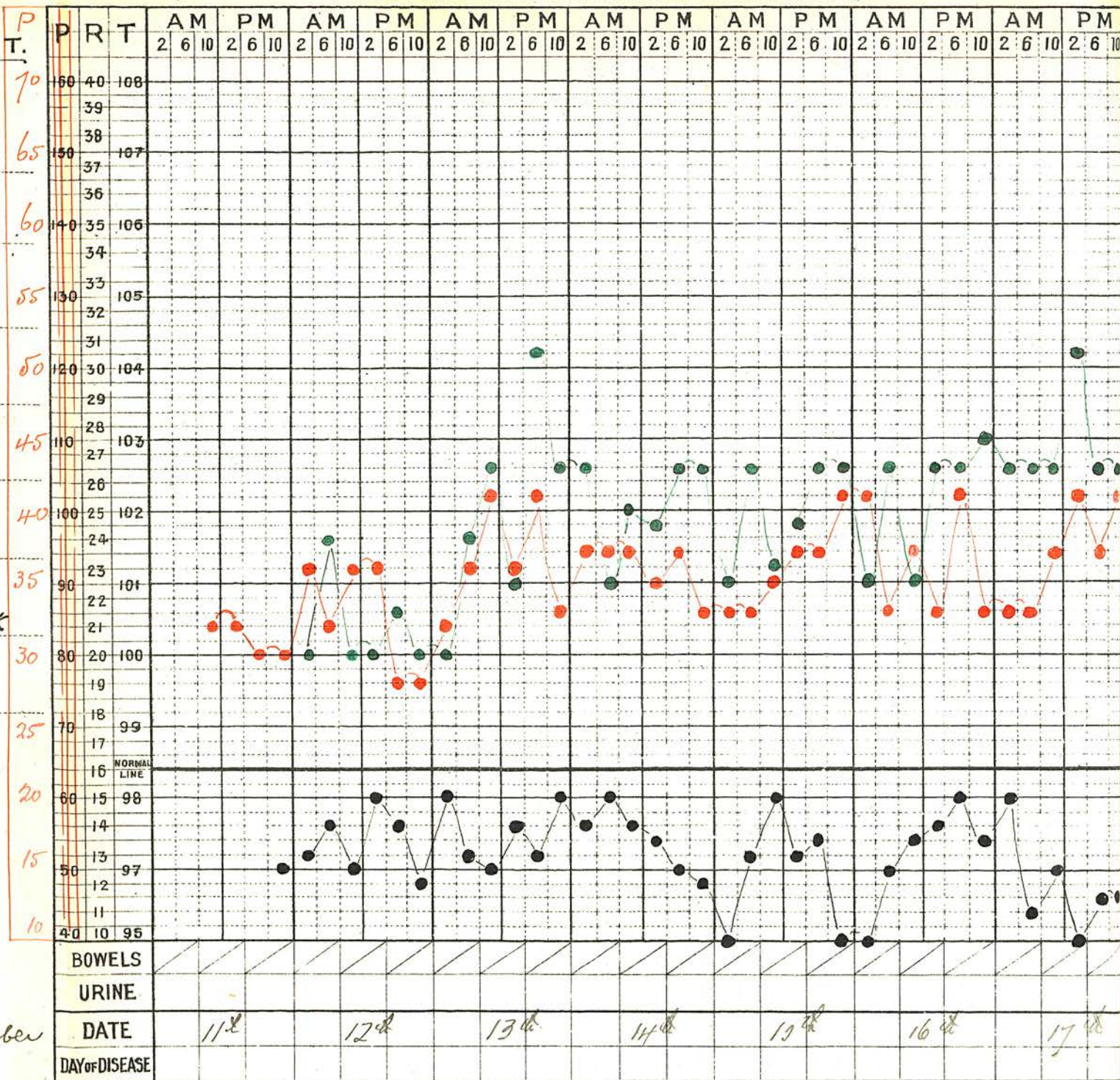
CASE 6.

Complete Heart-Block with History of Adams-Stokes Syndrome.

MATILDA MARSHALL, Aet. 63.

Date of admission.	December 10th, 1910.
Complaint.	Weakness and fainting.
General history.	Patient was married at the age of 27. She has had three miscarriages, and one live child who lived to the age of 14 years. She had a bad attack

RESULT Death
7th January,
1911



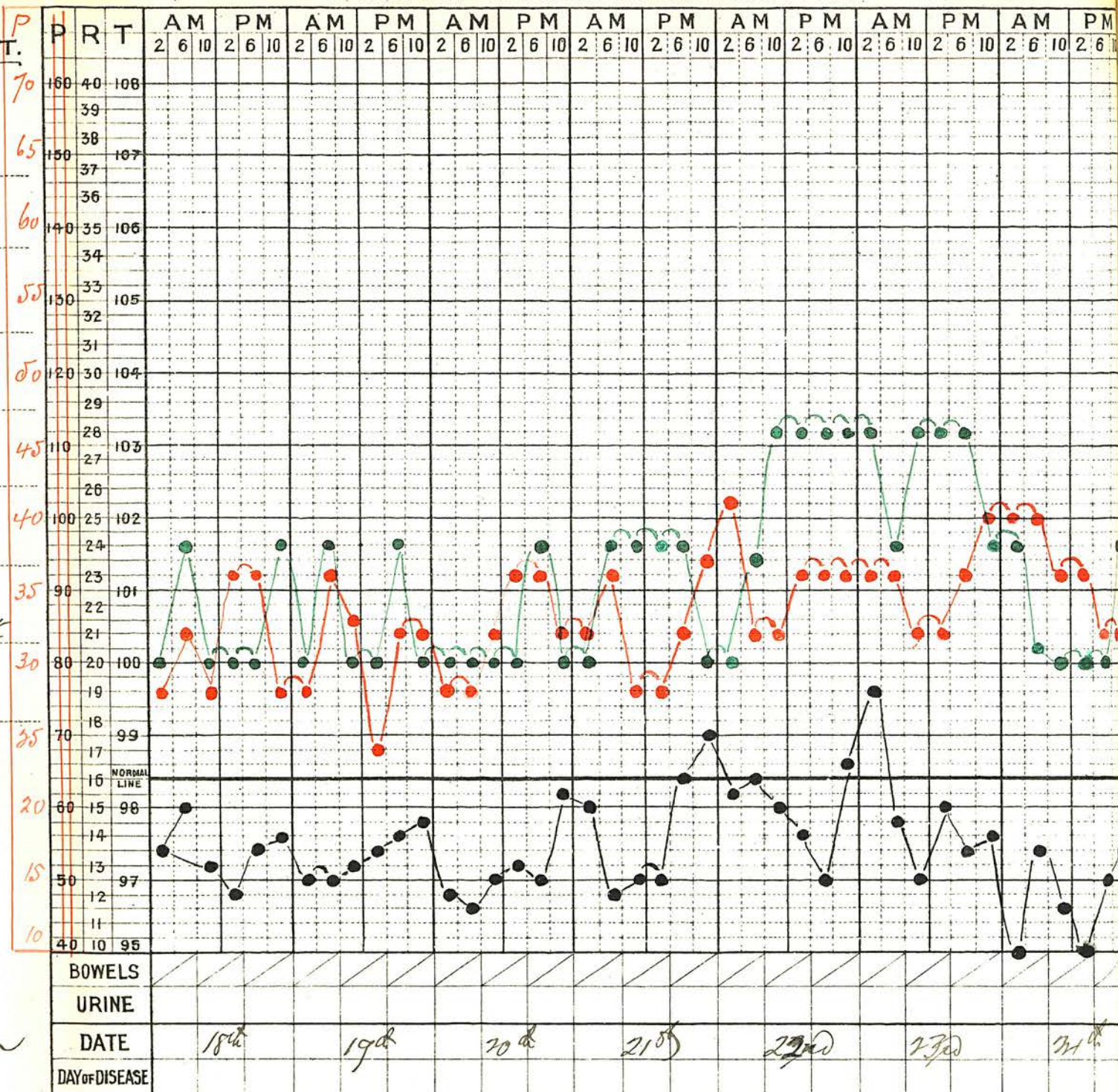
December

TEMPERATURE IN BLACK

No 2.

WALTON WORKHOUSE HOSPITAL.

4 HOURS CHART.

NAME *Matilda
Marshall*AGE *63 years*OCCUPATION *House-
wife*ADMISSION *11th Decr,
1910*DISEASE *Heart-block*RESULT *Death
7th January,
1911**December*

PULSE IN RED

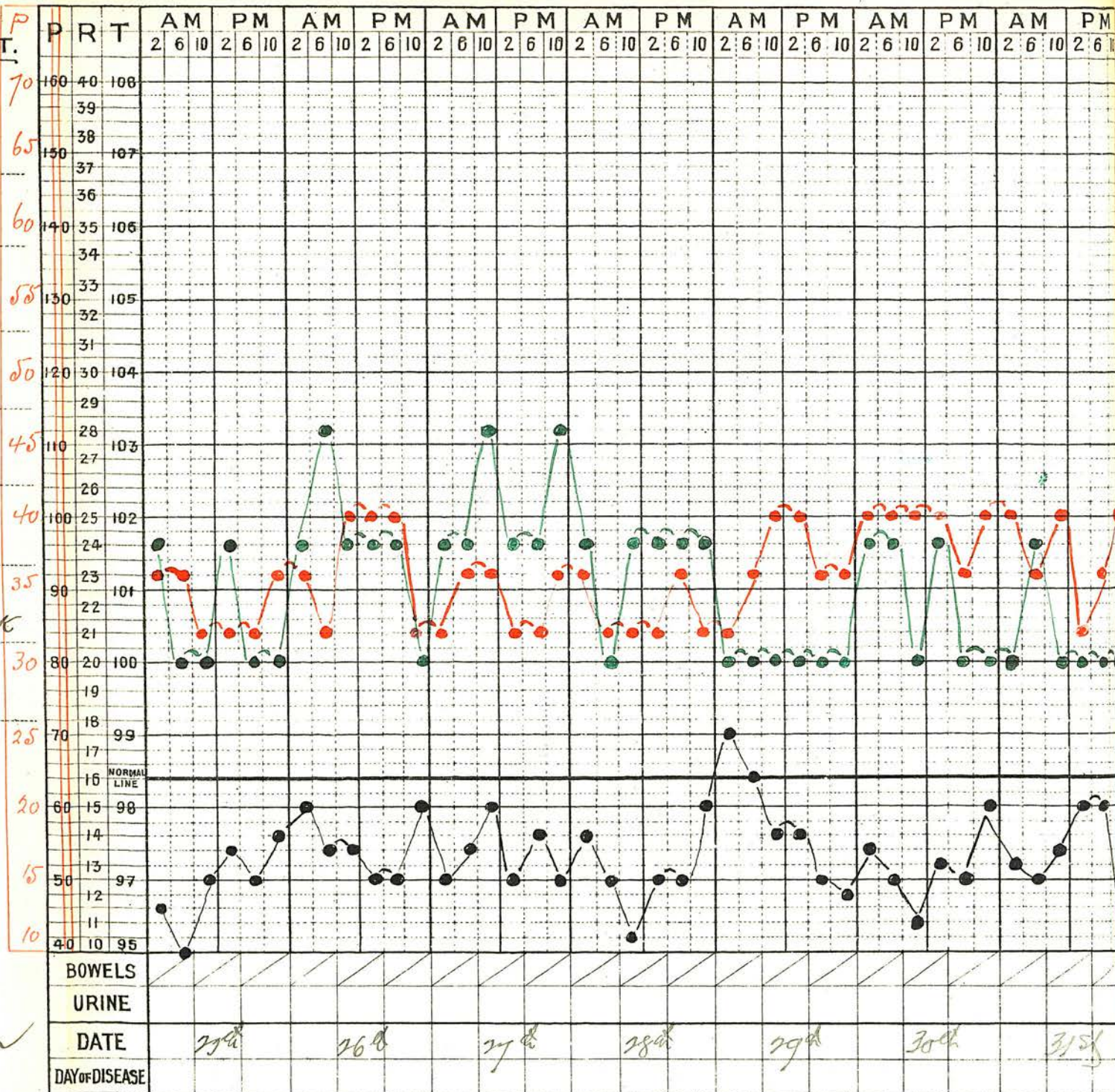
RESPIRATION IN GREEN

TEMPERATURE IN BLACK

No 3

WALTON WORKHOUSE HOSPITAL.

4 HOURS CHART.

NAME *Natilda
Marshall*AGE *63 years*OCCUPATION *House-
wife*ADMISSION *11th Decr,
1910.*DISEASE *Heart-block*RESULT *Death
7th January,
1911*

December

WALTON WORKHOUSE HOSPITAL.

4 HOURS CHART.

NAME *Natilda Marshall*

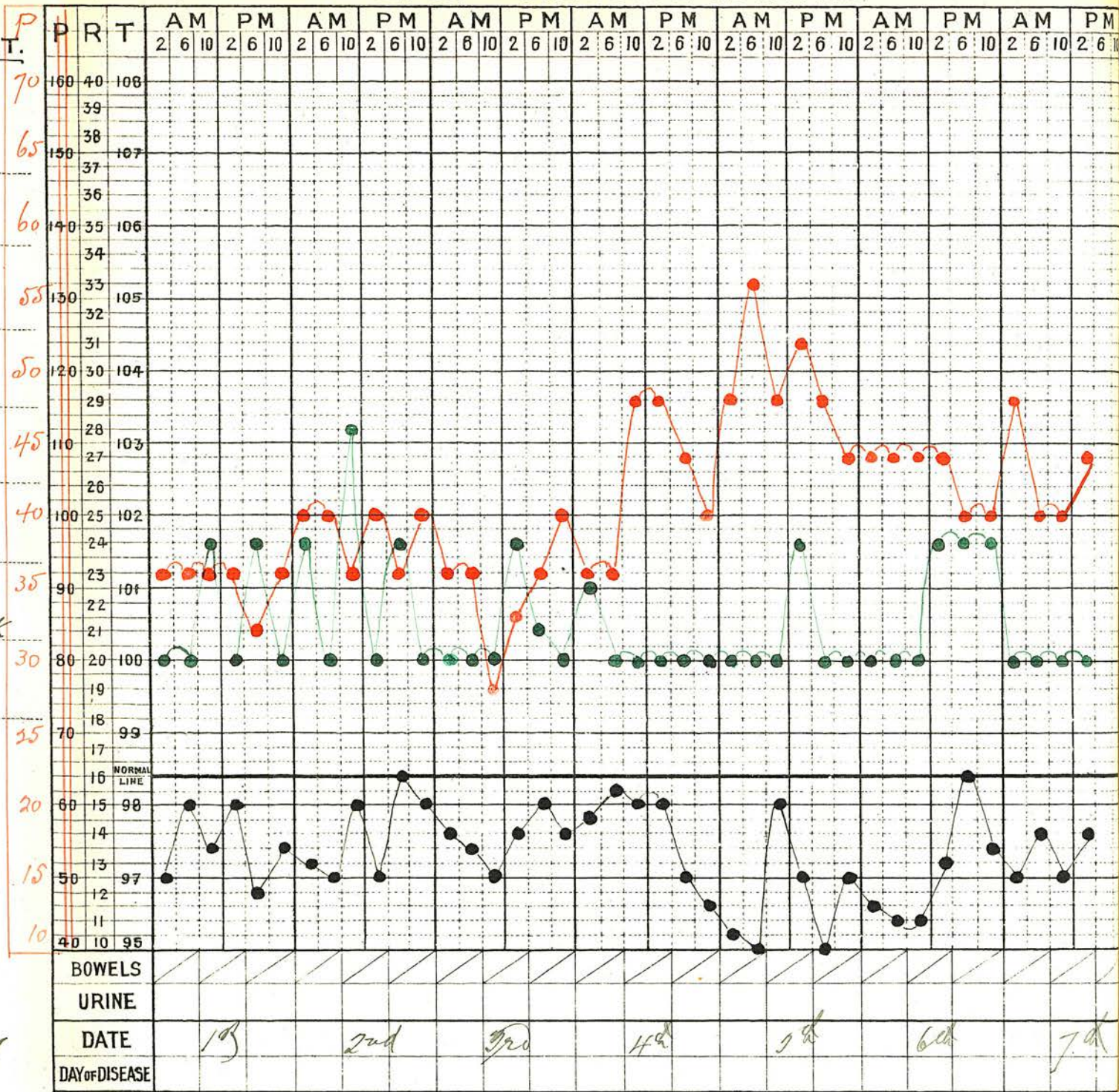
AGE *63 years*

OCCUPATION *Housewife*

ADMISSION *11th Decr. 1910*

DISEASE *Heart block*

RESULT *Death 1st January, 1911.*



PULSE IN RED

RESPIRATION IN GREEN

TEMPERATURE IN BLACK

of small-pox three years after marriage, and is markedly pitted. She has had no other illnesses, but has never been a robust woman.

In 1908 she was treated in Walton workhouse hospital for ulcers of the leg which were specific in nature, and in 1909 she was again treated for the same.

On August bank holiday, 1910, she became very nervous and fainted every time she moved. She became "dizzy in the head" and then lost consciousness. She came to the Walton workhouse hospital shortly afterwards. She had no attacks of faintness or giddiness whilst in hospital, but was not under the writer's observation at the time. She was discharged on the 25th September. Shortly after discharge she began to faint again and the slightest exertion was sufficient to bring on an attack. If she rose suddenly from a chair or went to the lavatory she was almost certain to have a fainting attack.

State on admission On re-admission on the 10th December, 1910, she was found to be a thin, anæmic-looking woman, and on examination she complained of abdominal pain in addition to weakness and fainting. Her temperature was 97, and respiration 30.

Circulatory system The rate of the pulse is 32, and it is regular in force and frequency. (*vide* pulse chart.) The systolic blood pressure is 290mm. of Hg., and the diastolic blood pressure 105mm. of Hg. The arteries are very thickened and tortuous. There are marked jugular pulsations in the neck, about three times as rapid as the apex beat impact; the apex beat is visible and palpable. Faint pulsations can also be felt at the level of the second and third left interspaces, presumably auricular in nature. The right border of the heart is $1\frac{1}{2}$ inches from mid-sternum and the left border is 4 inches from mid-sternum. There is a swishing systolic murmur at the apex propagated onwards into the axillary region. On auscultation over the second and third left interspaces extra sounds may be heard, one loud and long immediately after the first sound, and one short and sharp after the second sound. Pulsations corresponding in time to the extra sounds are visible in the jugular veins, and therefore it is presumed that these extra sounds represent auricular beats.

Nervous system. There is nothing abnormal to note.

Digestive system. Patient suffers from constipation.

Renal system. Urine, sp. gr. 1012. Reaction is acid, and there are no abnormal constituents.

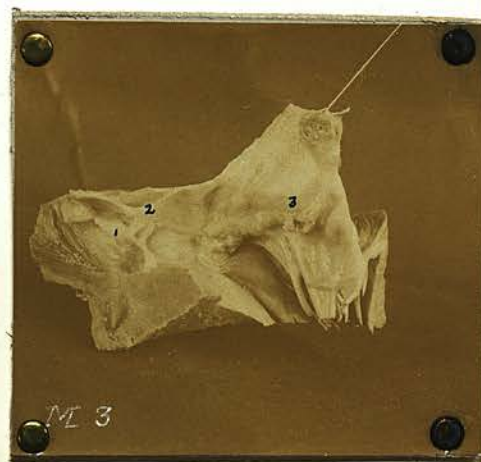
M. 1. Right Heart

1. Superior Vena cava
2. Annulus Ovalis
3. Falcia Terminalis
4. Marginal cusp of Tricuspid valve
5. Septal cusp of Tricuspid valve
6. Wall of Conus Arteriosus
7. Pulmonary Artery
8. Aorta
9. Mouth of Coronary Sinus

M 1

M. 2. Left Heart

1. Aorta. 2. Pulmonary Artery. 3. Wall of left Auricle
4. Left Coronary Artery. 5. Aortic cusp of Mitral Valve
6. Marginal cusp of Mitral Valve



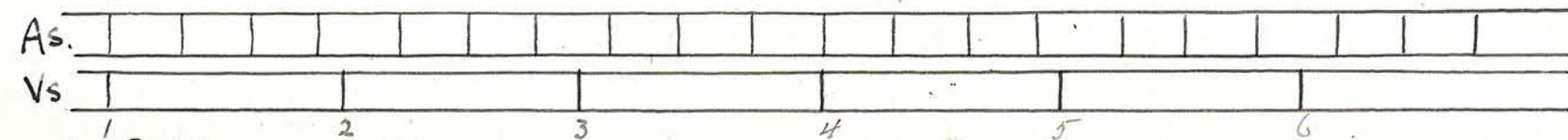
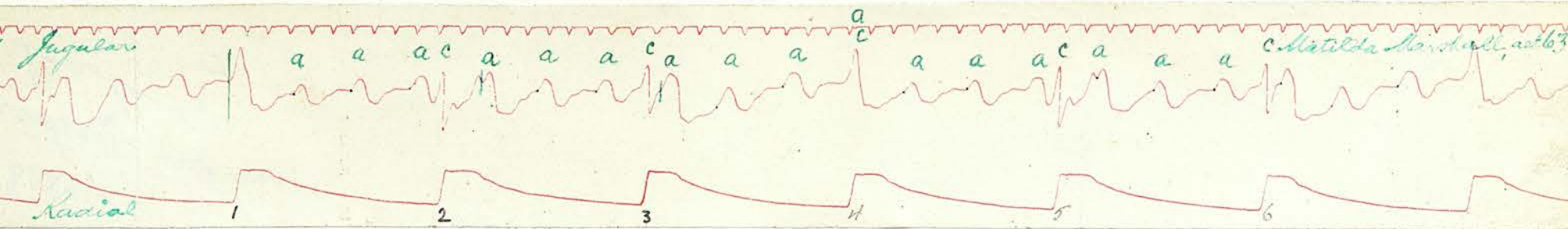
M. 3.

1. Anterior cusp of Aortic Valve
2. Right posterior cusp of Aortic Valve
3. Ventricular Surface of Aortic cusp of the Mitral Valve, showing the ridge of calcareous nodules passing from the cusp into the septal wall below the right posterior cusp of the Aortic Valve.

M 2

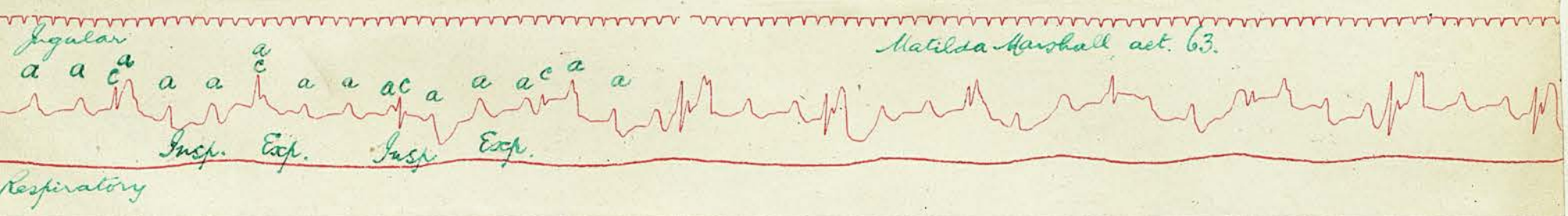
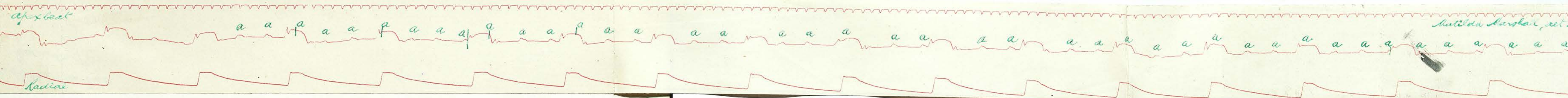
Matilda Marshall.

Matilda Marshall



As = Auricular Systole
Vs = Ventricular Systole

Complete Heart-block



Progress and treatment of the case.

Bowels were very constipated on admission. She was kept under observation until December 16th, the only treatment being an occasional dose of white mixture. She had no fits during this period.

Shortly after admission, tracings, which demonstrated complete heart-block, were taken. On December 16th she was put upon atropine gr. $\frac{1}{100}$ four-hourly, but there was no change in the conduction, auricles and ventricles still continuing to beat independently. Vagus stimulations (pressure) were tried without any result, and the patient was now ordered a potassium iodide mixture. Her breathing later became embarrassed, and she was put upon brandy, oz. $\frac{1}{2}$, strychnine gr. $\frac{1}{60}$ four-hourly. She gradually grew weaker, and the pulse was imperceptible on January 7th, 1911, and remained so until she died on January 9th.

She had no seizures or syncopal attacks whilst in hospital.

Graphic records.

The combined tracings show complete auriculo-ventricular heart-block.

Auricular frequency 105.

Ventricular frequency 30.

The diagram constructed underneath tracing No. 1 clearly indicates complete dissociation of the auricular and ventricular rhythms.

Tracing No. 2 shows combined apex and radial tracings, and tracing No. 3 shows jugular tracing and respiratory curve.

Pathological Examination of the Heart.

Heart.

The heart weighs 470 grammes.

External measurements.

Circumferential measurement in the long axis, 38.5 cm.; circumferential measurement around the auriculo-ventricular groove, 31 cm.; length 14 cm.; breadth 10.2 cm.; antero-posteriorly 8.0 cm.

Long diameter of the cavities.

Right auricle, 6.2 cm.; right ventricle, 5.4 cm.; left auricle, 6.5 cm.; left ventricle 7.2 cm.

Epicardium.

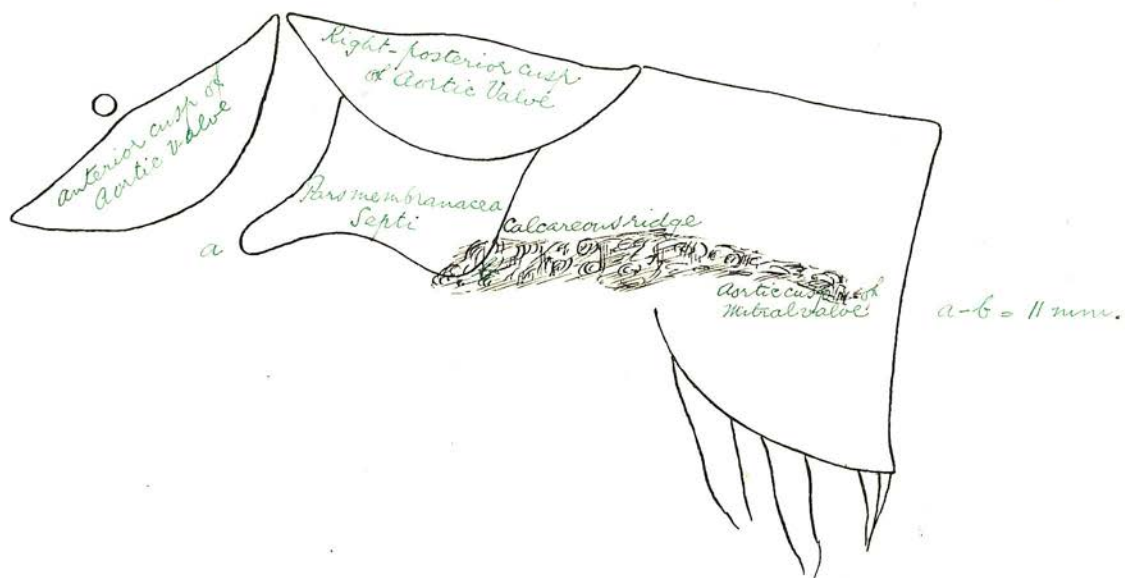
There is a considerable deposit of adipose tissue over the right side of the heart, especially over the basal part of the right ventricle and the conus arteriosus. There are small "milk-spots" over the anterior and outer aspects of the right ventricle.

Right auricle.

The endocardium shows slight diffuse thickening. The foramen ovale is closed. The musculature is hypertrophied, especially the *tænia terminalis*, the pectinate muscles and the annulus ovalis.

The auricular septal wall, felt from the right side immediately above the attachment of the septal cusp of the tricuspid valve, is firm and dense. No fibrous change in the region of the junction of the superior vena cava and auricle can be seen.

Matilda Marshall



The pars membranacea septi and
calcareous ridge upon the aortic
cusp of the mitral valve.

- Tricuspid orifice. The tricuspid orifice is 10cm. in circumference. The valve segments and chordæ tendineæ are normal, except for some thickening of the free margin of the marginal cusp. The papillary muscles are not fibrous.
- Right ventricle. The musculature is markedly hypertrophied, the muscle wall being in most places 6mm. thick. No fibrous change is visible.
- Pulmonary orifice. The pulmonary orifice is 7.0cm. in circumference. The pulmonary valve and artery are healthy.
- Left auricle. The endocardium is slightly thickened, and the musculature is somewhat hypertrophied.
- Mitral orifice. The mitral orifice is 8.0cm. in circumference. Both mitral segments are thick, shrunken, hard and infiltrated with calcareous deposits. The aortic cusp is the more affected of the two, and the calcareous masses project as bosses upon both the auricular and ventricular surfaces of this cusp. Projecting upon the ventricular surface of the aortic cusp, about mid-way between the attached margin and apex, there is a calcareous ridge which passes inwards to near the inner end of the line of attachment of the cusp, and passing thence in the septal wall towards the posterior angle and the lower side of the pars membranacea septi, terminates at a point 6mm. below the deepest point of the attached margin of the right posterior cusp of the aortic valve. The pars membranacea septi, although translucent is somewhat thickened and is of small size (see diagram.) The chordæ tendineæ of the mitral valve are short and thick. The papillary muscles are large and thick and present numerous small patches of fibrosis.
- Left ventricle. The musculature is hypertrophied. The muscle wall is 1.5cm. thick at the level of the apex of the posterior papillary muscle, and 1cm. thick at the apex of the cavity. Numerous fibrous areas are present in the ventricular muscle.
- Aortic orifice. The aortic orifice is 6.0cm. in circumference. The basal portions of all the cusps are thick, and the endocardium in the deepest part of each sinus of valsalva is thick and fibrous. Small atheromatous patches are present in the wall of the ascending aorta, and the wall of the coronary arteries are markedly atheromatous.

Naked Eye and Microscopic Examination of the Brain and Vagus Nerves.

- The Brain. The brain weighs 1396 grammes. Examination of the surface of the brain and of cut sections does not reveal the presence of any abnormality except, perhaps, a slight congestion of the vessels on the surface.

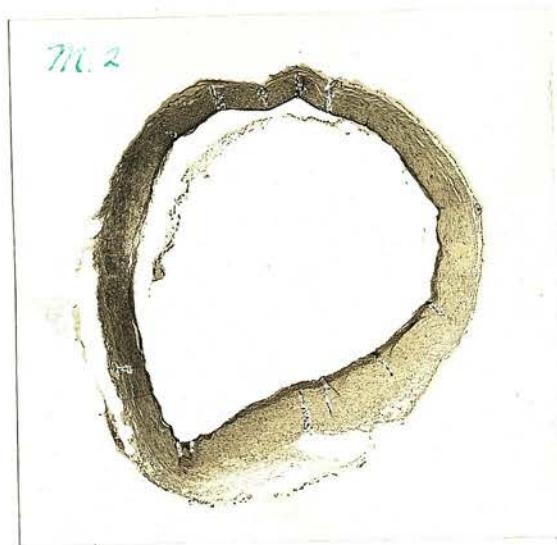
MATILDA MARSHALL



× 24 Diameters

M 1. Section through Medulla Oblongata at level
of the Vagal Nuclei.

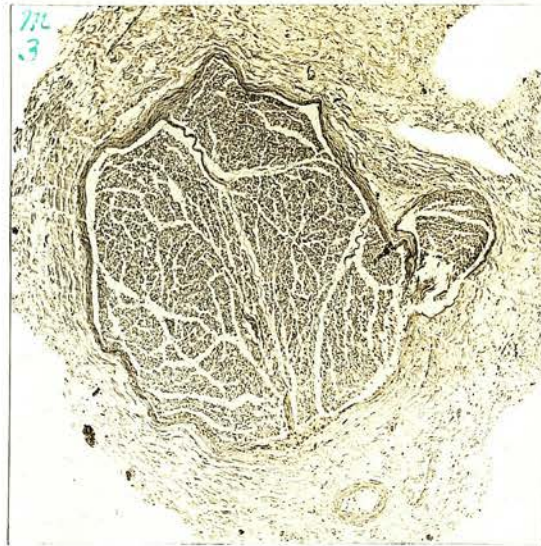
MATILDA MARSHALL



× 15 Diameters

M 2. Section of Basilar Artery.

MATILDA MARSHALL



× 38 Diameters

M 3. Transverse section of Vagus Nerve.

Sections show a practically normal condition of the vessels cut, the lumen being good and the walls not unduly thickened. There is no evidence of either endarteritis, thrombosis or atheroma. The middle coat is rather thicker upon one side of the artery than it is upon the other.

Medulla oblongata. Section through medulla oblongata at level of the vagal centres.

Vagal nuclei. The majority of the vagal nuclear cells are shrunken so as to appear to lie in widely dilated spaces, and within the cells themselves the nuclei are for the most part ill-defined and in some cases not visible at all. The intra-cellular structure is also ill-defined as regards the differentiation between granules and fibrils. There is no obvious small round-celled exudation into the pericellular lymphatic spaces, and none of the vessels show signs of thrombosis, though the smaller arterioles are distinctly thicker-walled than normal. It is difficult to say how much of the nuclear cell degeneration is due to post-mortem change, but it is noteworthy that here and there an almost normal nerve cell is to be seen from which one would infer that the degeneration of the others had occurred before death.

Vagus nerves. Transverse sections of the nerves. There is no small round-celled infiltration and no excess of fibrous tissue, but whereas many of the nerve fibres look normal, many others stain obscurely and have begun to break down into granular and fatty debris. Many spaces are seen from which the nerve fibres have disappeared altogether. The appearances are far from normal, and owing to some of the fibres looking almost natural, the changes in the others are probably ante-mortem.

CASE 7.

Complete Heart-Block, with History of Adams-Stokes Syndrome.

THERESA KRALL, Act. 88.

Date of admission. February 2nd, 1911.

Complaint. Debility.

General history. The history is difficult to take accurately, as the patient and her daughters are Jewesses and do not speak English well. Three years ago she became troubled in her health and had fainting attacks. Her daughter in broken English described one as follows:—Her eyes stared and then she fell down in a faint. When she was recovering she twisted her face, moved her hands, and made inarticulate noises.

When the seizures first commenced she had an attack almost every week, and usually had three or four seizures in a day. She then became less active in her habits, and the fits were not so frequent. During the

WALTON WORKHOUSE HOSPITAL.

4 HOURS CHART.

NAME *Theresa
Krahl*

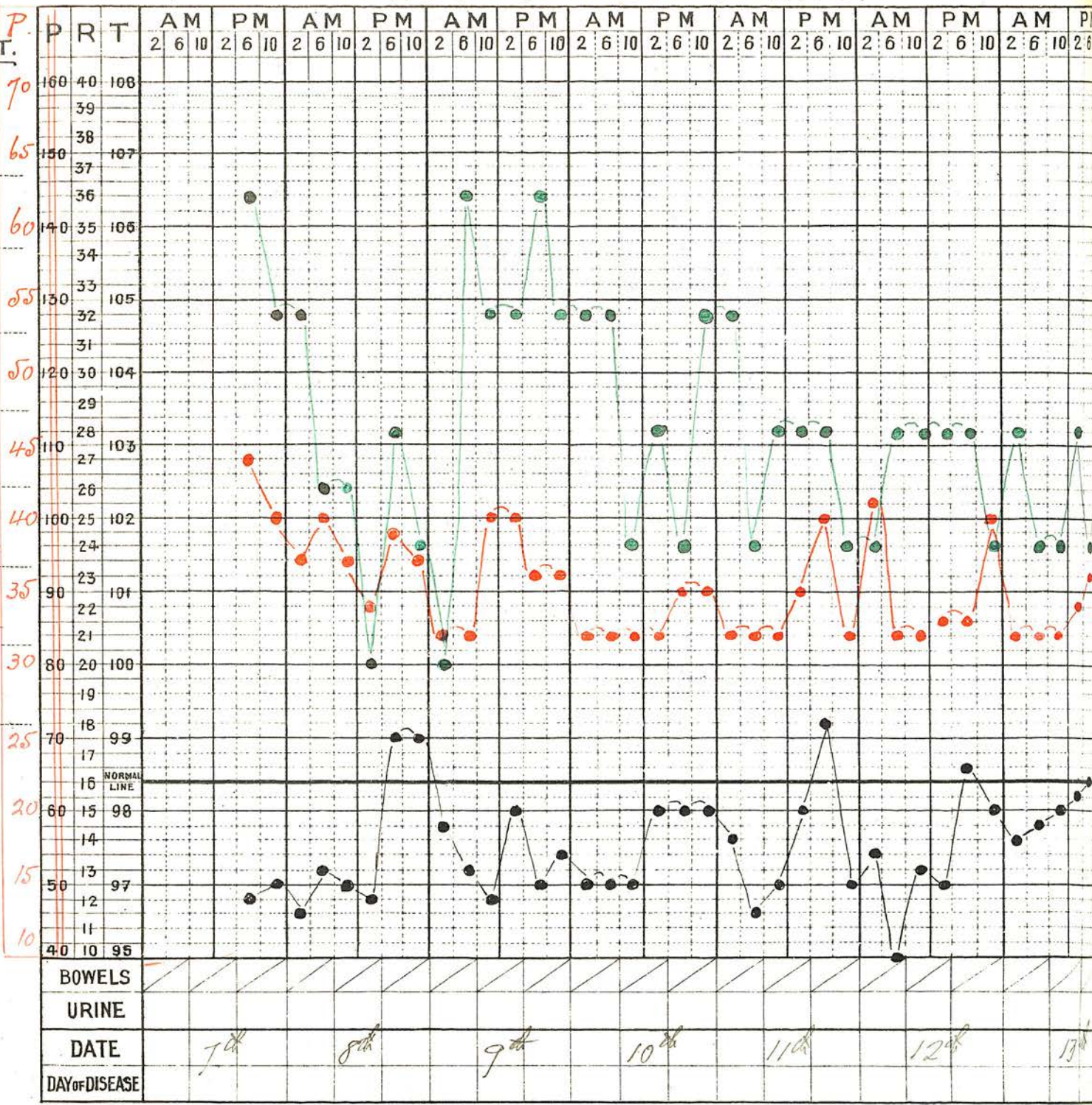
AGE *88 years*

OCCUPATION *house-
wife*

ADMISSION *7th Feb. 1910*

DISEASE *Heartblock*

RESULT *condition
unchanged*



PULSE IN RED RESPIRATION IN GREEN TEMPERATURE IN BLACK

P

70

6.

60

5

50

4

40

30

2.

20

17

WALTON WORKHOUSE HOSPITAL.

4 HOURS CHART.

NAME *Theresa Krall*

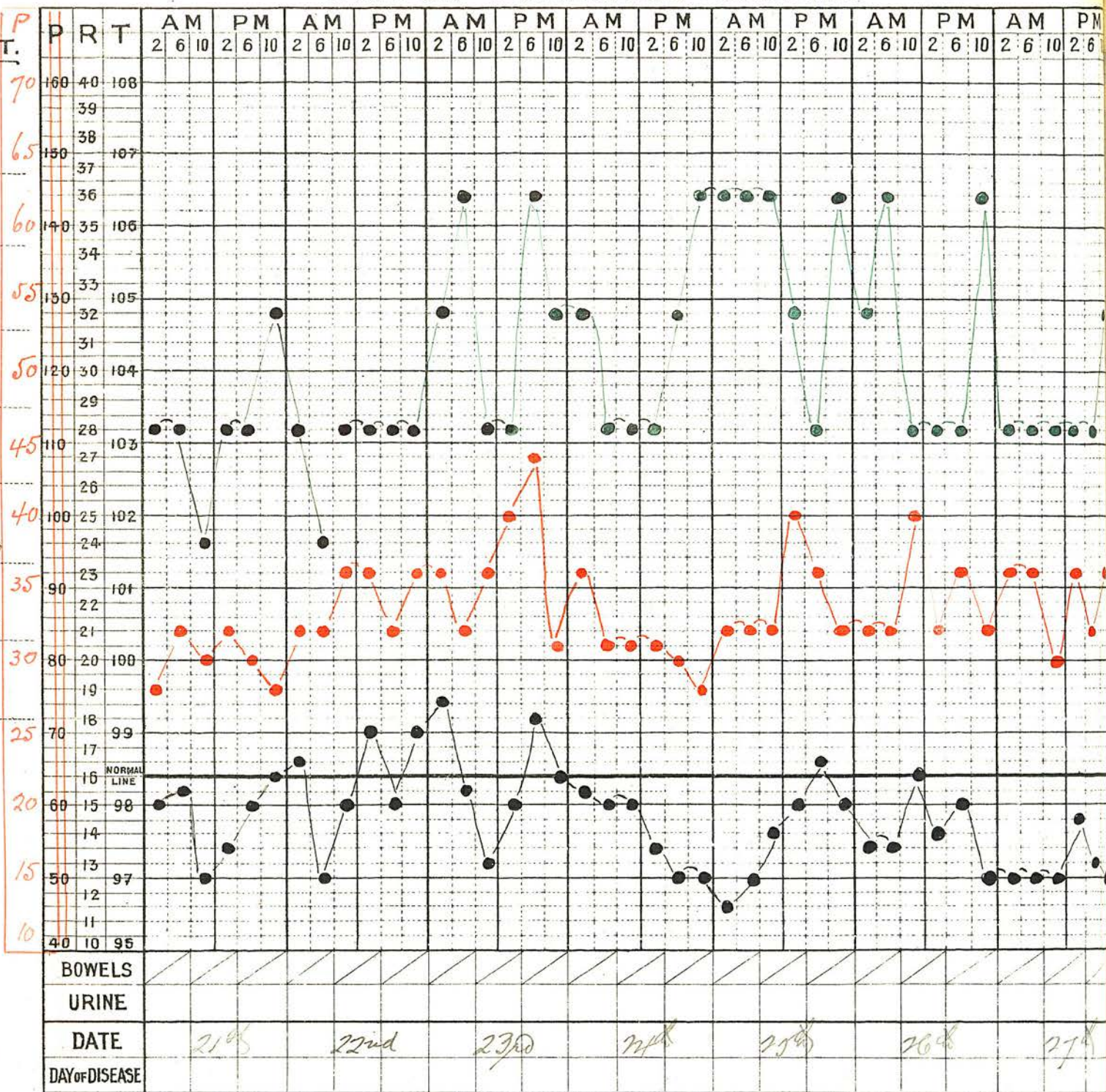
AGE *88 years*

OCCUPATION *Housewife*

ADMISSION *7th Feb. 1910*

DISEASE *Heart block*

RESULT *Condition unchanged*



PULSE IN RED RESPIRATION IN GREEN TEMPERATURE IN BLACK

WALTON WORKHOUSE HOSPITAL.

4 HOURS CHART.

NAME *Theresa Krall*

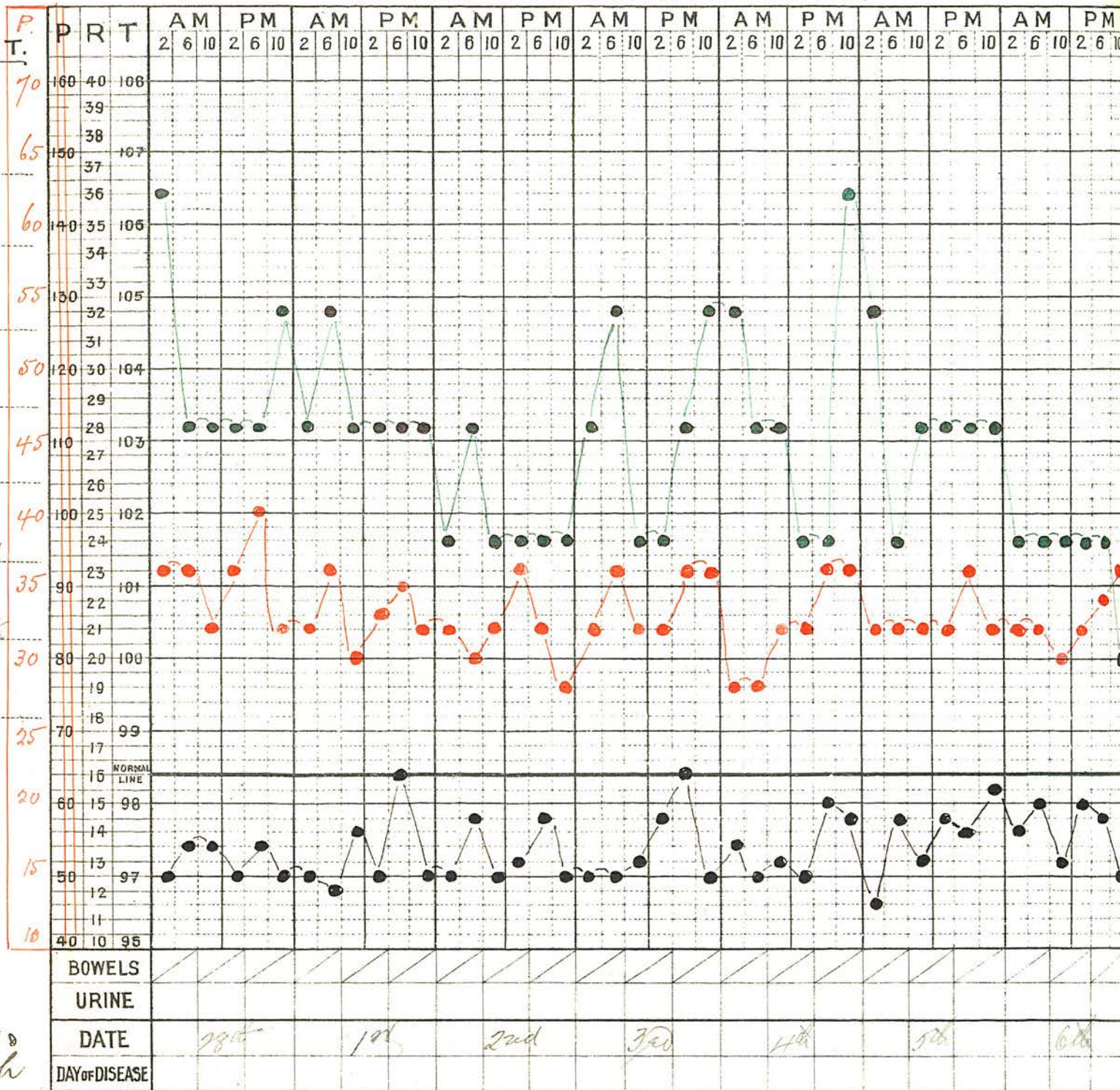
AGE *88 years*

OCCUPATION *Housewife*

ADMISSION *7th Feb. 1911*

DISEASE *Heart block*

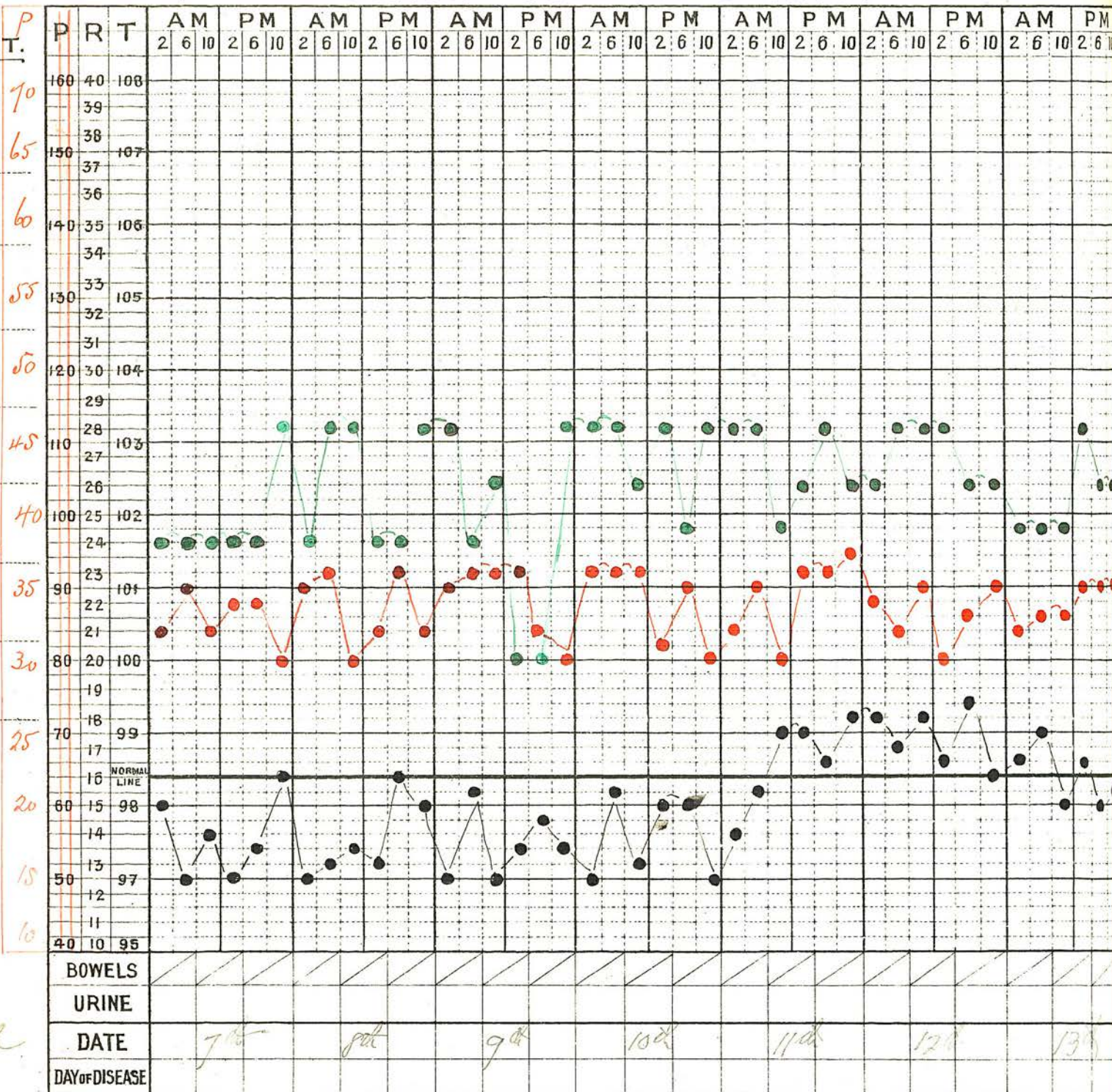
RESULT *Condition unchanged*



*Feb 8
March*

WALTON WORKHOUSE HOSPITAL.

4 HOURS CHART.



March

WALTON WORKHOUSE HOSPITAL.

4 HOURS CHART.

NAME *Theresa Krall*

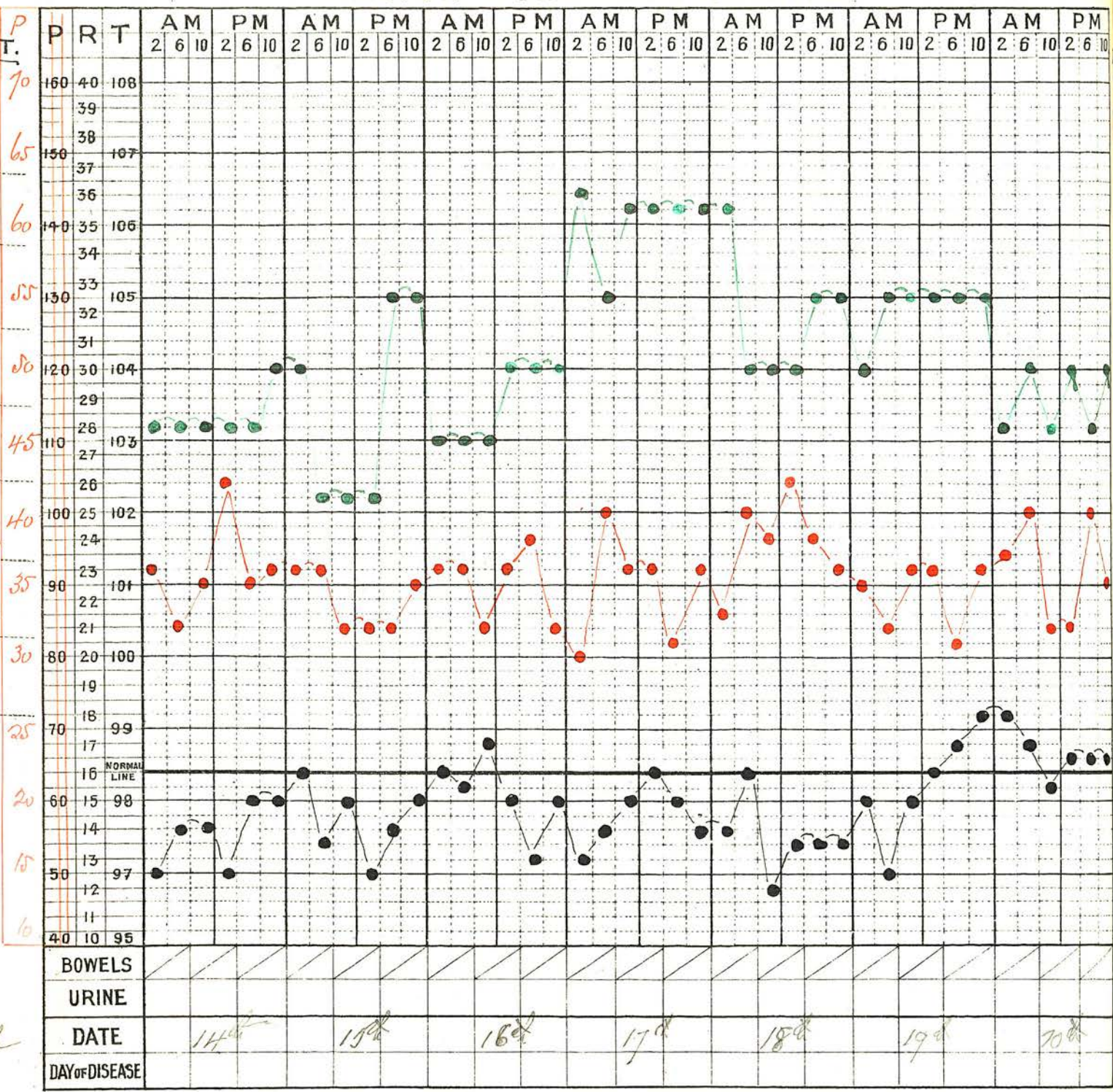
AGE *88 years*

OCCUPATION *Housewife*

ADMISSION *7th Feb. 1910*

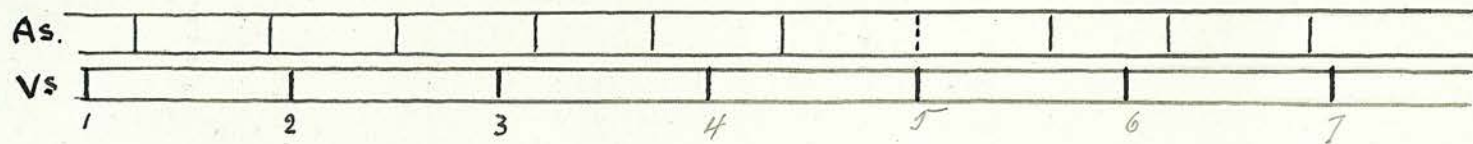
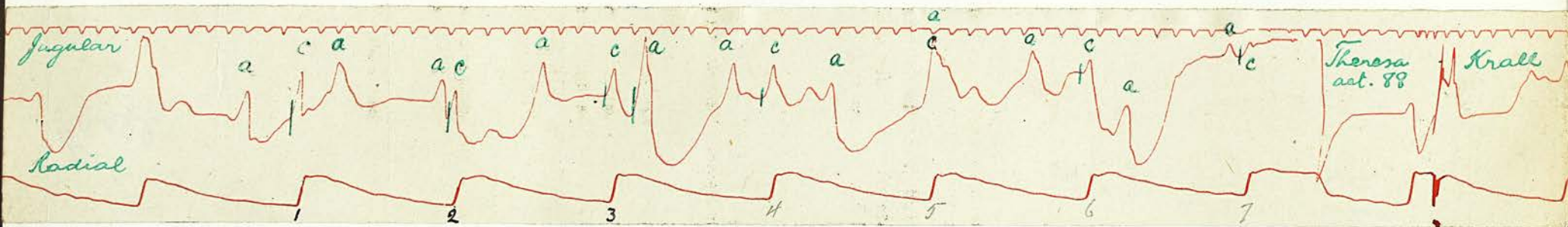
DISEASE *Heart block*

RESULT *Condition unchanged*



PULSE IN RED RESPIRATION IN GREEN TEMPERATURE IN BLACK

Theresa Krall



As. = Auricular Systole
Vs. = Ventricular Systole

Complete Heart-block.

last year the attacks have come about every three months, and four weeks ago she had two seizures in the same day. Between the attacks she enjoyed good health. She has always been a healthy woman and has had no serious illnesses.

State on admission The patient is a bright intelligent old woman, but is somewhat emaciated and suffers from anæmia.

Circulatory system Jugular pulsations are visible in the neck and are about twice as rapid as the radial pulse; the apex beat is not palpable; there is a soft systolic mitral murmur propagated into the axillary region; there are no audible auricular sounds, and the first sound in the aortic region is roughened.

The right border of the heart is $1\frac{1}{2}$ inches to the right of mid-sternum, and the left border is 4 inches to the left of mid-sternum.

The pulse varies between 32 and 40 and is regular in force and frequency (*vide* pulse chart). The arteries are thickened. The maximum systolic blood pressure is 210 to 220mm. of Hg., and the diastolic blood pressure is 98 to 100mm. of Hg.

Nervous system. There is nothing of importance to note.

Respiratory system. The lungs are emphysematous.

Alimentary system The tongue is clean and the bowels are regular; the liver is slightly enlarged.

Renal system. Urine, sp. gr. 1025. Reaction is acid, and there is no albumen.

Progress and treatment of the case. Shortly after admission tracings were taken demonstrating on analysis the existence of complete heart-block. Stimulations of the vagus (pressure) entirely failed to affect the frequency of the ventricles. The vagi were paralyzed by the administration of atropine gr. $\frac{1}{100}$ four-hourly, but the block did not pass off.

The patient is at present on tonic and supporting treatment, and is fairly well. There is no change in the circulatory system. She has not had any epileptiform or syncopal attacks since admission.

Graphic records. Tracings demonstrate complete auriculo-ventricular heart-block.

Auricular frequency 60.

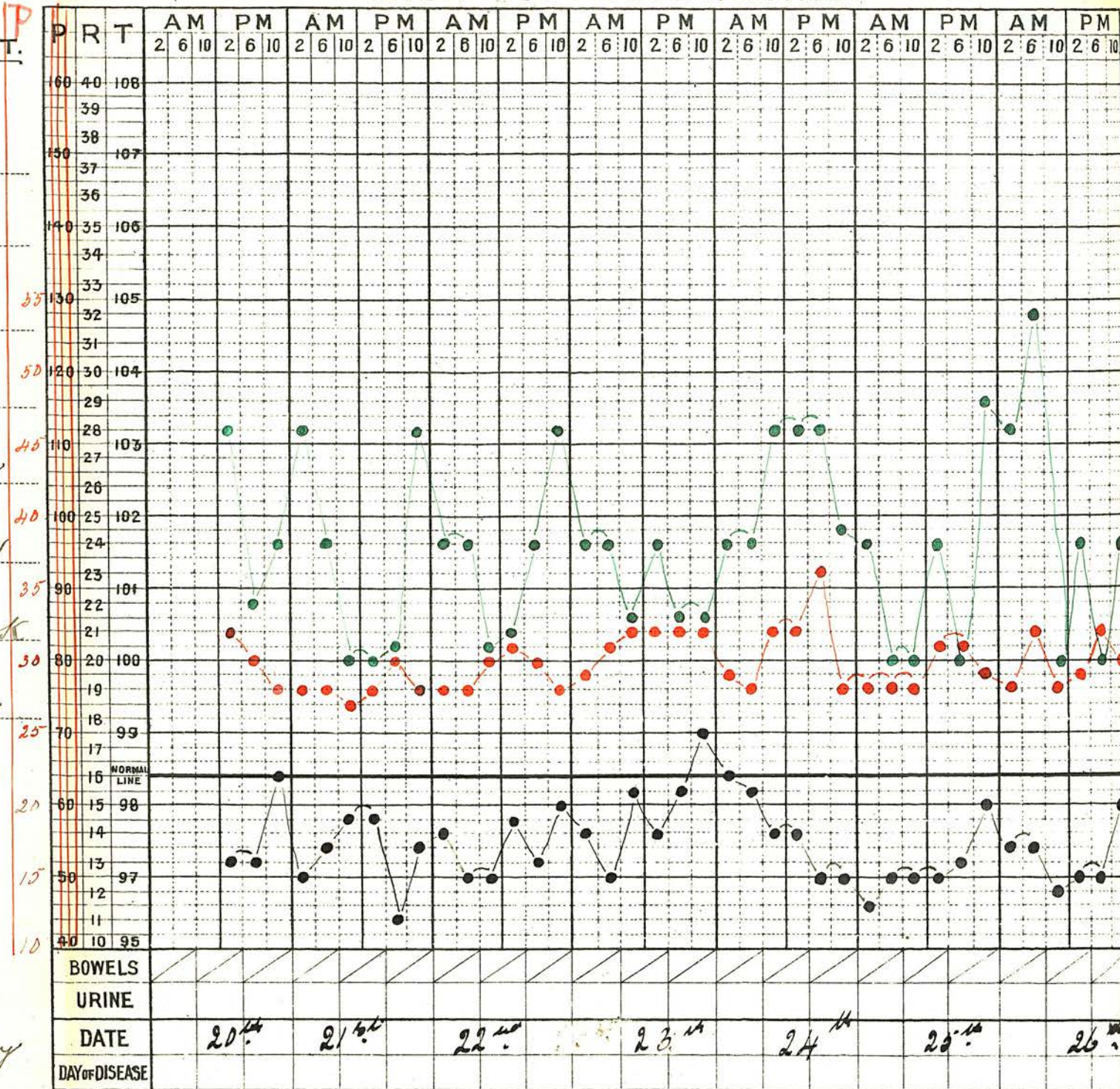
Ventricular frequency 37.5.

The schematic analysis shows that the auricles and ventricles are beating altogether independently.

No 1

WALTON WORKHOUSE HOSPITAL.

4 HOURS CHART.

NAME *Jones**Nelson*AGE *60 years*OCCUPATION *Railway*ADMISSION *20 Feb. 1911*DISEASE *Heart Block*RESULT *No change**February*

No 2

WALTON WORKHOUSE HOSPITAL.

4 HOURS CHART.

NAME

James
Nelson

AGE

60 years

OCCUPATION

Railway
Porter

ADMISSION

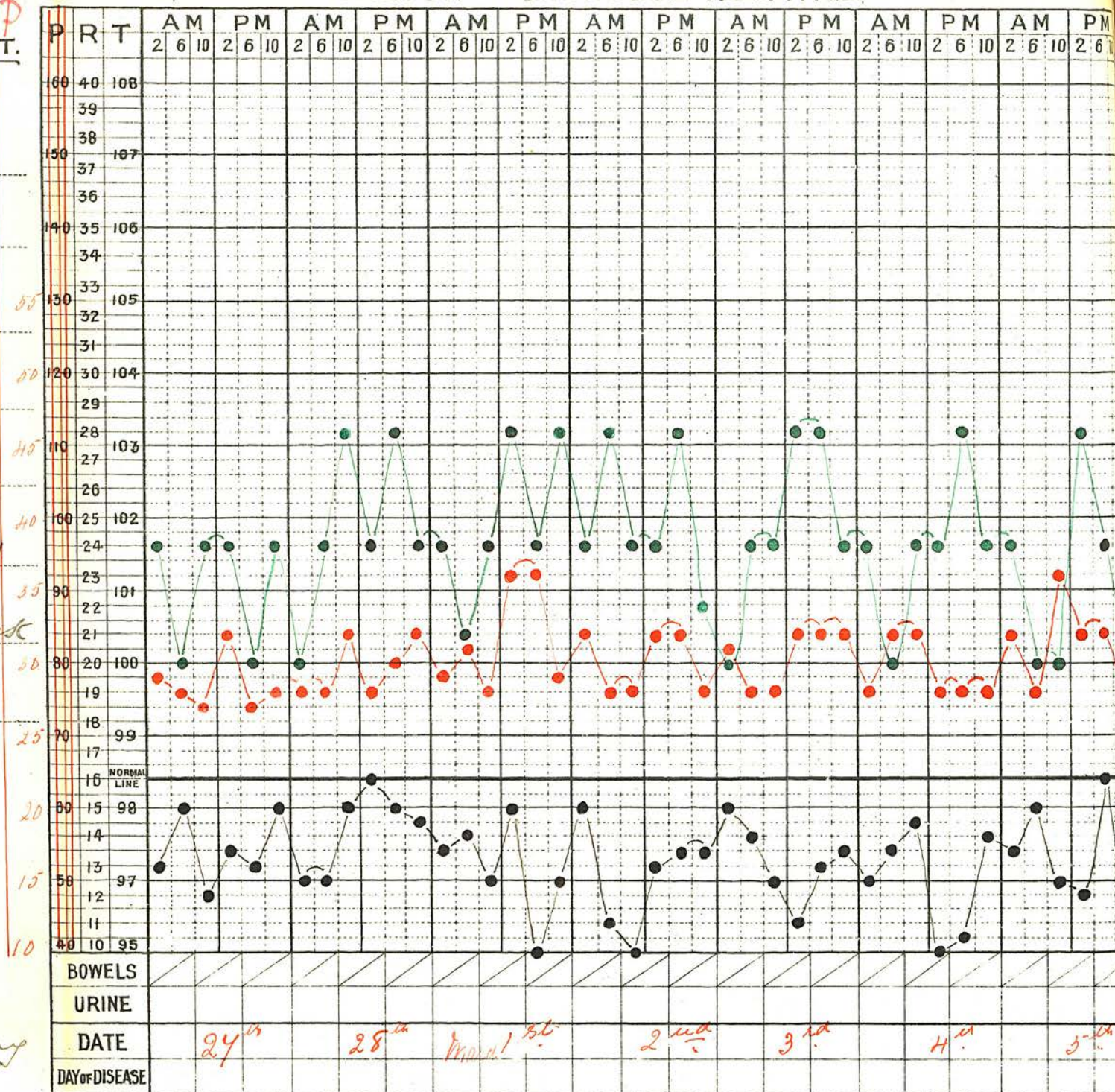
20 Feb. 1911

DISEASE

Heart Block

RESULT

No change



PULSE IN RED

RESPIRATION IN GREEN

TEMPERATURE IN BLACK

No 3.

WALTON WORKHOUSE HOSPITAL.

4 HOURS CHART.

NAME

James
Nelson

AGE

60 years

OCCUPATION

Railway
Porter

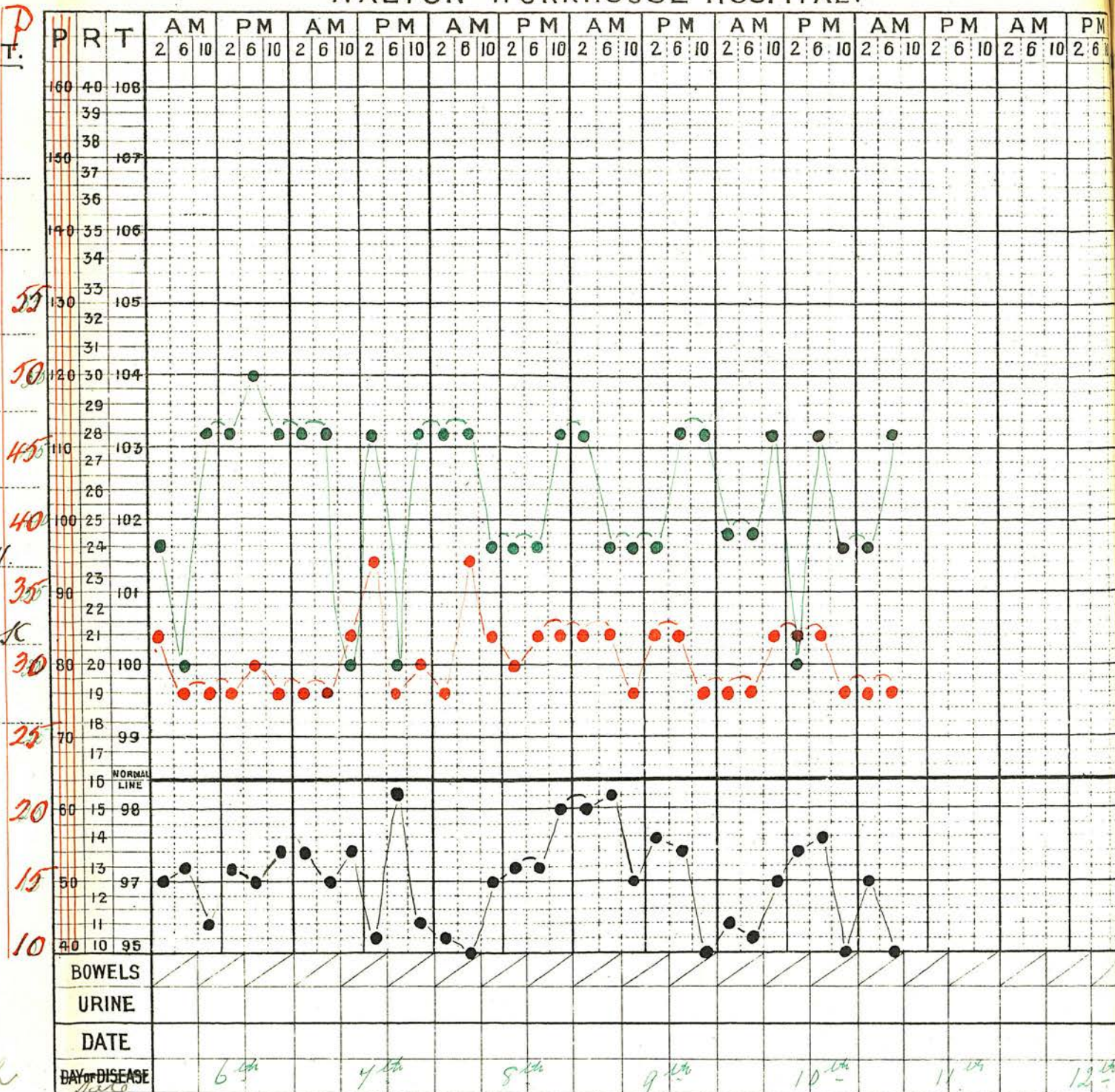
ADMISSION

20 Feb. 1911.

DISEASE

Heart Block

RESULT

Discharged
10th March, 1911.
No change.

PULSE IN RED

RESPIRATION IN GREEN

TEMPERATURE IN BLACK

CASE 8.

Complete Heart-Block with History of Adams-Stokes Syndrome.

JAMES NELSON, Aet. 60.

Date of admission. February 20th, 1911.

Complaint. Giddiness and weakness.

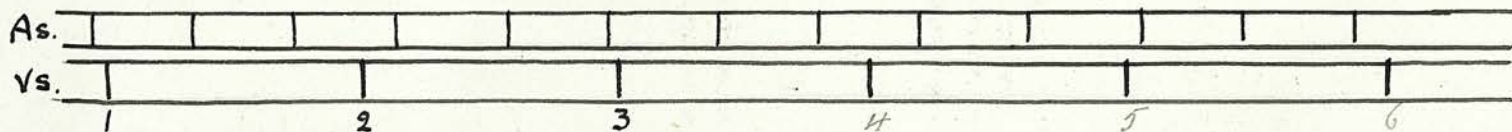
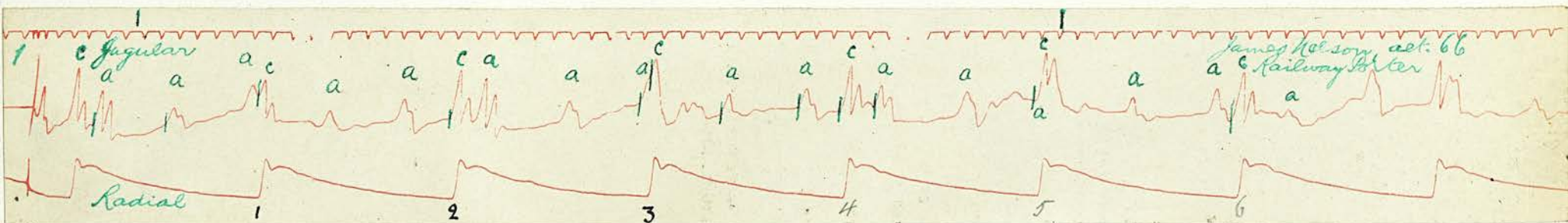
General history. Patient states that as a child he suffered from scarlet fever, small-pox and measles. He commenced life as an iron-moulder, but did not stay long at this employment. He then went to sea as a steward. He gave up sea life and became a barman in a public-house. For the last 26 years he has acted as a railway porter. His work has always been hard. He has taken an average amount of alcohol and has smoked to excess. There is no history of syphilis or of rheumatism. Three years ago he had to forsake his work because of "swoons." At first he did not lose consciousness during the attacks but a "weakness came over him." He suffered from these attacks for about six months, when his health improved and they ceased, and for the following eighteen months he enjoyed fairly good health. The attacks then recommenced and became more severe. His wife told him that when the attacks came on he lost consciousness, dropped suddenly wherever he happened to be, waved his arms, jerked about and did not recover consciousness for about two minutes. He has had attacks in the street and has fallen and injured himself. During the last four months he has had to the best of his recollection seven attacks.

State on admission He is a healthy looking man, and states that he feels pretty well now, and has only come to hospital "for a few days' rest."

Circulatory system The arteries are thickened, but not tortuous. The systolic blood pressure is high, being equal to 200mm. of Hg., and the diastolic blood pressure is equal to 90mm. of Hg. The rate of the pulse is 28 per minute, and it is perfectly regular (*vide* pulse chart). Faint pulsations are visible in the external jugular vein and are more frequent than the carotid waves. The heart has a forcible beat in the fifth left intercostal space, just outside the mammary line. The lateral borders of the heart are $1\frac{1}{4}$ inches to the right of mid-sternum and $5\frac{1}{2}$ inches to the left of mid-sternum. Auscultation reveals a short mitral systolic murmur propagated into the axillary region. There is no basic murmur. The second aortic sound is clear and distinct, and there is an occasional reduplication.

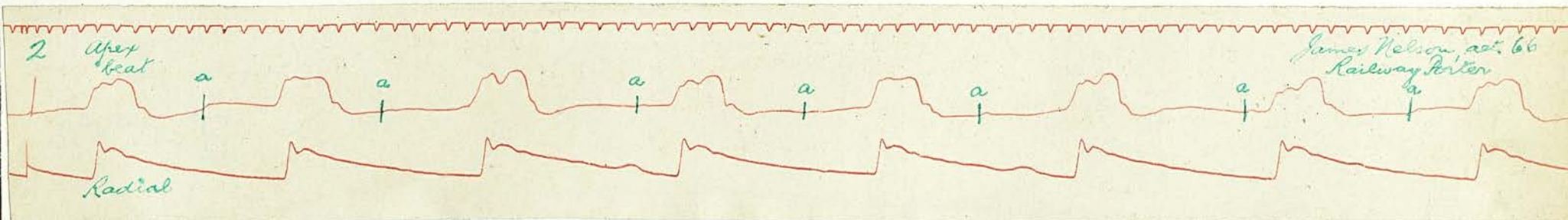
Nervous system. There are no obvious symptoms, except the giddiness and faintness complained of.

James Nelson



As. = Auricular Systole
Vs. = Ventricular Systole

Complete heart-block



Respiratory system. There is emphysema of the lungs, and a few crepitations at both bases.

Digestive system. The appetite is good and there is slight increase in the size of the liver.

Renal system. Urine, sp. gr. 1010. Reaction is neutral, and there is a slight trace of albumen.

Progress and treatment of the case. Tracings were taken shortly after admission demonstrating complete heart-block. On the 28th February, atropine in doses of gr. $\frac{1}{60}$ was administered four-hourly. There was no change in the conduction and atropine was discontinued. Vagus stimulations (pressure) were tried without any result. As will be seen from the pulse chart the pulse varied between 28 and 32.

The patient walked about quietly during his stay in hospital, was always in fairly good health, and had no seizures. On 11th March he requested to be and was discharged from hospital. He has been examined regularly since discharge, and his condition remains exactly the same.

Graphic records. The records show complete auriculo-ventricular heart-block.

Auricular frequency 67·5.

Ventricular frequency 30·0.

The auricular and ventricular rhythms are wholly independent of each other as is clearly demonstrated in the diagram under tracing No. 1.

No. 2 is a graphic record of apex beat and radial.

CHAPTER XIII.

Diagnosis.

- Pulse.** The pulse is always slow, rarely going above 40, and when the heart-block is complete there are remarkably few variations in its frequency. Before the onset of a syncopal attack the pulse disappears.
- The circulatory system.** There are visible pulsations in the jugular veins pursuing a regular rhythm which is more rapid than the ventricular rate. At the sternal margin of the 2nd and 3rd left interspaces light sounds may be heard (the beating of the left auricle), and shocks corresponding to these sounds may be seen or felt at the apex, and may be felt by carefully palpating the radial pulse.
- Graphic records.** The most satisfactory and sometimes the only method of diagnosis is by taking simultaneous tracings of the jugular and radial pulse, or jugular and apex beat, and analyzing them. By this method the a.c. interval may be measured and depression of conductivity noted, and the degree of heart-block (an occasional dropping of a ventricular systole, a 2-1, 3-1, or 4-1 rhythm, or complete block), may be demonstrated. If tracings are taken whilst the patient is having syncopal attacks the relationship of the auricular and ventricular frequency to the seizures may be seen.
- Fluorescopy.** Ritchie first used the fluorescent screen, and in 1905 (in the *Edinburgh Medical Journal*), described how pulsations of the auricles could be seen without any corresponding movement on the part of the ventricles.
- Electro cardiogram.** Gibson, Einthoven, Lewis, and others have demonstrated the existence of heart-block by the electro-cardiogram.
- Differential diagnosis.** The slow pulse of the old, of athletes, and that following infectious diseases often make tracings absolutely necessary, as the condition is often a true Bradycardia without any degree of heart-block.

In neurogenous cases the Bradycardia is due to vagal influence causing the heart-block, and this can be removed by the exhibition of atropine.

CHAPTER XIV.

Prognosis.

A prolonged a.c. interval or an occasional dropping of a ventricular systole is not of grave import; a patient may live for years and not be troubled in anyway, but such a patient should be kept under careful supervision. This condition strongly contra-indicates the exhibition of the digitalis group, as these drugs may aggravate the heart-block.

Toxic group.

Bradycardia may follow any of the acute infections, and then usually only lasts a few days.

The toxic group is very important. There is little doubt that many of the sudden deaths in diphtheria and pneumonia are due to heart-block, though in most cases the block is partial and completely clears up. In some of these post-febrile cases the bundle may be involved in an acute mural endocarditis, and they usually end favourably if the patient is kept at rest during the acute period.

Specific group.

Syphilitic cases are hopeful. Osler and Erlanger observed and recorded a case with loss of consciousness and slow pulse. The heart-block was complete. Under iodide of potassium he recovered quickly. The cerebral and cardiac symptoms disappeared and his heart returned to normal.

Arterio-sclerotic group.

The arterio-sclerotic group are unaffected by treatment. Their course is fairly slow, the majority of the cases living from 5 to 6 years, and cases have been recorded which lasted twenty years. There is no known drug which improves conductivity or prevents stoppage of the ventricles.

The syncopal attacks make life uncertain, as death so often occurs in a seizure. Where the fits are numerous the prognosis is bad, as in case 1 of the writer's series, where the patient had over 150 seizures in one day. Case 2 suffered from the disease for 3 years, whereas in case 7 and 8 the disease has lasted over three years, and the patients are still alive. Cases of complete block are often capable of a considerable amount of work. No. 8 of the series is at present doing light work, and beyond his slow pulse there is nothing to indicate anything abnormal. Death is often hastened by a complicating bronchitis and œdema of the lungs, as in Nos. 3, 4 and 5 of the series.

Many die in their first attack and the condition remains undiagnosed.

Gerhardt reports a case in which heart-block cleared up owing to cessation of the pathological process in a bundle not totally obliterated.

In the intervals between the seizures, the patient is often remarkably well, and when the syncopal and epileptiform attacks disappear (usually when the block becomes complete) the patient may be very comfortable

for years. The heart is capable of wonderful efficiency with a pulse rate under 40. Frequently recurring syncopal attacks with prolonged stoppage of the ventricles are always dangerous.

Dyspnœa, cough and œdema of the lungs occur in many cases.

CHAPTER XV.

Treatment.

Considering the pathological change producing these cases it must be said that in the majority of them it is useless to give remedies to cure.

The diet should be plain and the patient should lead a quiet, uneventful life, avoiding any sudden exertion or strain, as the area of cardiac response is seriously impaired. The bowels should be regular and all straining at stool avoided. Easy going and simple diet should be the rule of life.

Syphilitic cases are hopeful, and the administration of large doses of iodide of potassium may cause the absorption of the syphilitic infiltration. One must not forget the possibilities of Salvarsan in cases with an undoubted syphilitic history.

Of the host of drugs used, strychnine seems to be the most favourable, and it is possible that fibrolysin may be of some use.

Digitalis is dangerous, especially in partial heart-block, as it increases the depression of conductivity. Where the block is complete and the heart is failing, with signs of dilatation, the digitalis group might be exhibited with benefit.

In the cases following pneumonia and influenza the prognosis is hopeful, and rest in bed and tonic treatment are all that is required to cause a subsidence of the inflammatory process.

Atropine is often given to eliminate any neurogenous element. Gibson records a case where the administration of atropine caused a complete block to disappear temporarily. There is a possibility however, when the block is partial, that atropine may precipitate a complete block by increasing the auricular frequency.

In complete block the ventricles are dissociated from the auricles, and anything affecting the auricular rate is not of importance. The patient may enjoy gentle exercise, avoiding lifting, straining, and holding his breath, as the attacks seem to be due to c.o._2 acting on the ventricular muscle wall.

If the blood pressure is high the nitrites may be exhibited.

The usual restorative measures may be employed during an attack, and, where there is a tendency to the occurrence of syncopal attacks the patient should not run the risk of their occurring in dangerous situations.

CHAPTER XVI.

Summary and Conclusions.

General.

In presenting eight cases of heart-block, observed within a period of eighteen months, certain points of interest are suggested.

The senile form of heart-block is probably more common than is generally supposed, especially among people who have worked hard and lived recklessly. Alcoholism, arduous employments, gonorrhœa, syphilis, rheumatism and smoking seem to be prominent etiological factors. In looking over old hospital records such remarks as "senile epilepsy," "epilepsy with slow pulse," and "vertigo" make one suspect that the condition was heart-block with Adams-Stokes Syndrome. Men are more often affected than women. In the writer's series five were men and three were women, but the large majority of cases reported in the literature of heart-block have been of the male sex. All the cases of the series had reached the arterio-sclerotic period; six were between the ages of 60 and 65, one was between 65 and 70, and one was 88 years of age.

Weakness, giddiness, "mazy bouts" (a colloquialism, meaning attacks of giddiness), are usually the first symptoms of the condition.

It is interesting to note that between the cerebral attacks there are periods of comparative comfort.

After the preliminary attacks the pulse may rise to its normal rate, and the patient may apparently recover entirely; and further, when the pulse rate falls to the uniformly slow rate of complete heart-block there is rarely any disturbance of consciousness. Cerebral attacks, therefore, usually occur when the heart-block is partial; when the heart-block becomes complete they are much less likely to happen. This was emphasised by Gibson and Ritchie in the case of Sir William Gairdner, and is seen in some of the cases under consideration.

Complete heart-block may be complicated by most grave and possibly lethal attacks of syncope or epileptiform seizures, as in the case of Thomas Alcock.

Atropine may be of value in preventing the passage of reflex influences down the vagus which might precipitate seizures in a patient in whom the block is partial. In complete heart-block atropine has no effect on the ventricular frequency.

The digitalis group should be used with considerable caution because of their property of increasing the depression of conductivity ; but, when the block is complete and the myocardium shows definite signs of failure with dilatation, digitalis might be exhibited with benefit.

The cases at present under consideration differ from the experience of others in that they seem to be of a more severe type, living for a much shorter period and exhibiting very extensive degenerative changes, (post-mortem).

In the majority of cases the block is associated with atheroma of the coronary vessels and interstitial myocarditis, causing degenerative changes in the node and a.v. bundle, and therefore, one cannot hope for much improvement by the exhibition of drugs.

It is very desirable that tracings should be taken to complete the chain of evidence and that fallacies should be avoided in interpreting them.

THOMAS ALCOCK.

Thomas Alcock.

Thomas Alcock is an example of one of those rare cases with complete severance of the bundle, and yet with Adams-Stokes seizures.

The calcareous degeneration of the musclic ring around the mitral orifice is of importance. Is this the site of greatest strain? The calcareous rings in this case, and in the cases of Green and Campbell, are certainly *not* thrombosed and then calcified vessels.

The non-coronary cusp of the aortic valves lies in close relationship to the left septal branch of the bundle, and therefore the relationship of the calcareous mass to the right posterior cusp is of significance (see diagram).

The microscopic examination of the bundle demonstrates complete severance of the bundle.

The tracings are full of interesting features. In the first place analysis shows complete dissociation of the auricular and ventricular rhythms.

C.M.=Convulsive Movements.

Thomas Alcock.

Chart to show the relationship of the Auricular and Ventricular frequency to the Seizures.

There seems to be a distinct relationship between the auricular and ventricular frequencies and the convulsive seizures. Some time before a fit one finds the auricle beating every $\frac{3}{5}$ of a second, and the time between the consecutive ventricular systoles may be 13-fifths. The auricle gradually slows down until it is beating every $5\frac{1}{2}$ or 6-fifths of a second, and at the same time the ventricle gradually accelerates until it is beating at about the same rate as the auricle, though quite independently, as a careful analysis of the record shows. This rate precedes the seizure. The ventricle stops, and this stop is followed by rapid increase in auricular frequency. The rigidity and convulsive efforts begin about 13 or 14 seconds after ventricular stoppage. After the seizure the ventricle beats about every 5 seconds, whilst the auricle beats with great frequency. The same cycle of events happens again. The ventricular rate increases and the auricle slows down until its frequency approximates to that of the ventricle, though with entire dissociation of rhythms, and then another seizure takes place. This same sequence of events happens time after time (see pulse chart).

The seizure F, in Record 4, shows interesting and distinctive features. After fit E the ventricle beats more quickly, each ventricular cycle being only 9 or 10-fifths of a second. After 10 such contractions there was a period of ventricular stoppage of 11 seconds only, and there was no rigidity or convulsions during or after this phase, as the cerebral anaemia was not sufficiently prolonged. Preceding the seizure F the auricular frequency had not slowed down to anything like the same extent as seen in the other records.

It is difficult to say what the relationship is between the auricular rate and the shortness of the periods of ventricular stoppage.

A ventricular stoppage of over 13 seconds seems to be associated with rigidity and convulsions.

What is the cause of ventricular stoppage and the seizures in this case where there is complete severance of the bundle? The preliminary retardation of the auricle points to vagus stimulation, but why should this affect the dissociated ventricles? The tracings do not afford any evidence that the seizures were brought on by a paroxysmal attack of marked ventricular tachycardia,—a sort of fluttering of the ventricles,—as described in one or two cases, and said to be due to irritation of the bundle below the lesion. There does not seem to be any satisfactory explanation.

Vagus stimulations, (pressure), and atropine were tried without any result. A careful examination of the central nervous system and the vagus nerves revealed nothing of importance.

This, therefore, is a case of complete auriculo-ventricular heart-block of purely myogenic origin, and yet with Adams-Stokes Syndrome.

EDGAR GREEN.

Edgar Green.

Edgar Green, like Alcock, is an example of complete auriculo-ventricular heart-block of the degenerative atheromatous type. His history of having five or six seizures at a time, and then being completely free from them for some time is instructive. He differs from Alcock in that when the block became complete the seizures ceased.

The tracings demonstrate complete auriculo-ventricular heart-block.

Atropine and vagus stimulations, (pressure), did not alter the frequency of the ventricular beat.

There is naked eye evidence of extensive calcareous change right through the region of the a.v. node and bundle. The basal portions of the mitral cusps are thick and calcareous. The aortic cusps are thickened, and the walls of the coronary arteries and their branches are atheromatous. In the basal part of the ventricle—at the site of the greatest strain?—there is a large horse-shoe shaped calcareous ring lying in the plane of the mitral orifice, (see diagram and X-ray photograph). Anteriorly, the ring commences just below the deepest part of the left posterior cusp of the aortic valve. It passes round close under, but does not involve the aortic cusp of the mitral valve, and thence passes round in the ventricular musculature close under, and in places even involving, the marginal cusp of the mitral valve. Thence it passes through the septum obliquely from left to right, and the posterior end projects into the lower part of the pars membranacea septi below the points of junction of the anterior and right posterior cusps of the aortic valve, (see diagram). The ring terminates in close relationship to the septal cusp of the tricuspid valve, and this septal cusp lies directly over the main bundle. In this case, as compared with the case of Frank Campbell, the ring passes further to the right, and comes more round between the right posterior and anterior aortic cusps, whereas in Campbell the ring stops short behind and under the right posterior cusp, (compare X-ray photographs and diagrams).

It may be said with certainty that this is a case of complete heart-block, with almost complete, or, at any rate, very extensive and marked involvement of the bundle.

THOMAS YATES.

Thomas Yates.

The tracings of this case demonstrate complete auriculo-ventricular heart-block.

The mitral valve segments present slight diffuse thickening and small yellowish fibrous areas in their substance, but they are not calcareous. The pars membranacea septi is a small translucent area—the sides of which measure 11mm., 11mm., and 4mm. respectively—below the right posterior cusp and the junction of this cusp with the anterior cusp of the aortic valve. The anterior and left posterior cusps of the aortic valve are slightly thickened, and the central portion of the right posterior cusp is calcareous. The endocardium in all the sinuses of valsalva is thick and fibrous. The coronary arteries are atheromatous.

There is no naked eye evidence of involvement of the bundle. A microscopic lesion may be present.

This case presents complete auriculo-ventricular heart-block without lesion of the a.v. bundle recognisable to the naked eye.

MARY RILEY.

Mary Riley.

The records of this case on analysis in one part show a condition resembling partial heart-block, whilst at another part the block seems to be complete. In the first part at 2 the a.c. interval is normal, and at 3 it is much prolonged, whereas the next auricular systole does not evoke a ventricular response, and this is maintained until the end of the tracing, where at X the beats are almost superimposed and at Y the a.c. interval is much diminished. The latter part then rather resembles complete heart-block.

The clinical evidence suggests partial heart-block.

There is no naked eye evidence of an extensive severing of the bundle, and if lesions are present at all they must be microscopic only.

The pars membranacea septi is a small relatively opaque triangular area, the sides of which measure 6, 4, and 7mm. respectively (see diagram). There is a considerable amount of fibrous change in the muscle wall of the left ventricle. The anterior and right posterior aortic cusps are slightly thickened, whilst the left posterior cusp is calcareous. The aorta presents nodular plaques of atheroma and some syphilitic cicatrisation of the wall. The coronary arteries show calcareous patches of atheroma.

Mary Riley is a case of partial, (complete ?), heart-block, with no naked-eye evidence of involvement of the bundle.

FRANK CAMPBELL.

Frank Campbell.

It was impossible to take graphic records in this case, but the general clinical evidence suggests a condition of partial heart-block.

The bundle is almost certainly involved by calcareous deposits, but even to the naked eye there is less certain proof of complete severance than in Green, as a comparison of the X-ray photographs and diagrams in both cases will show. The marginal cusp of the mitral valve is thick and shrunken, and in the muscle at the line of attachment of this cusp, at what is probably the site of greatest strain, there is a calcareous ring similar to that in Alcock and Green. This ring differs from that in Green in that it stops short behind and under the right posterior cusp of the aortic valve. Seen from the right side the pars membranacea septi is thick and semi-opaque, and of triangular form, the posterior side measuring 5mm., the lower side 6mm. and the anterior side 8mm. The coronary arteries are atheromatous.

The general evidence indicates that this is a case of partial heart-block with extensive and marked involvement of the bundle.

MATILDA MARSHALL.

Matilda Marshall.

The records of this case demonstrate on analysis complete auriculo-ventricular heart-block. The atropine test and vagus stimulations (pressure) were tried without any result.

Permission to examine the brain was obtained in this case, and a careful examination revealed nothing of importance.

The mitral segments are thick, shrunken, hard and infiltrated with calcareous deposits. The aortic cusp is the more affected of the two, and the calcareous masses project as bosses upon both the auricular and ventricular surfaces of this cusp. Projecting upon the ventricular surface of the aortic cusp, about mid-way between the attached margin and apex, there is a calcareous ridge, which passes inwards to near the inner end of the line of attachment of the cusp, and passing thence in the septal wall towards the posterior angle and the lower side of the pars membranacea septi, terminates at a point 6mm. below the deepest point of the attached margin of the right posterior cusp of the aortic valve (see diagram). The pars membranacea septi, although translucent, is somewhat thickened and of small size. Numerous fibrous areas are present in the ventricular muscle. The basal portions of all the aortic cusps are thick, and the endocardium in the deepest part of each sinus of Valsalva is thick and fibrous. The coronary arteries are markedly atheromatous.

Section of the wall of the left ventricle shows chronic interstitial myocarditis and a little fatty degeneration of the muscle fibres. The naked eye evidence suggests a complete block, and the condition appears to be an old inflammatory mitral lesion, in contrast to Alcock, Green and Campbell, where the changes are rather chronic, degenerative and atheromatous.

Marshall is a case of complete auriculo-ventricular heart-block with naked eye evidence suggesting a complete, or at any rate, a very extensive and marked involvement of the bundle.

Krall and Nelson. Tracings in Krall and Nelson demonstrate complete heart-block. The atropine test did not alter the ventricular frequency, and vagus stimulations (pressure), were tried without any result. Both patients are alive and well.

As far as the examination of the bundles has gone the following table indicates what may with certainty be said about the cases:—

CASE.	TRACINGS.	LESIONS.	REMARKS.
THOMAS ALCOCK ...	Complete heart-block.	Complete severance of the bundle, the result of degenerative atheromatous change (microscopically).	Presence of Adams-Stokes Syndrome and complete heart-block.
EDGAR GREEN ...	Complete heart-block.	Very marked and extensive involvement of the bundle area by degenerative atheromatous change (naked eye).	
THOMAS YATES ...	Complete heart-block.	No naked eye involvement of the bundle.	
MARY RILEY ...	Partial (complete?) heart-block.	No extensive severing lesion. If lesion at all must be microscopic.	Clinical evidence suggests partial heart-block.
FRANK CAMPBELL ...	No tracings.	Very marked and extensive involvement of bundle area by degenerative atheromatous change (naked eye).	Clinical evidence suggests partial heart-block.
MATILDA MARSHALL	Complete heart-block.	Bundle involved by old inflammatory mitral lesion (naked eye).	
THERESA KRALL ...	Complete heart-block.		Alive.
JAMES NELSON ...	Complete heart-block.		Alive.

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